Delmar HEALTH CARE

Clinical Application of MECHANICAL VENTILATION

Fourth Edition

David W. Chang

Clinical Application of
MECHANICAL VENTILATION

David W. Chang

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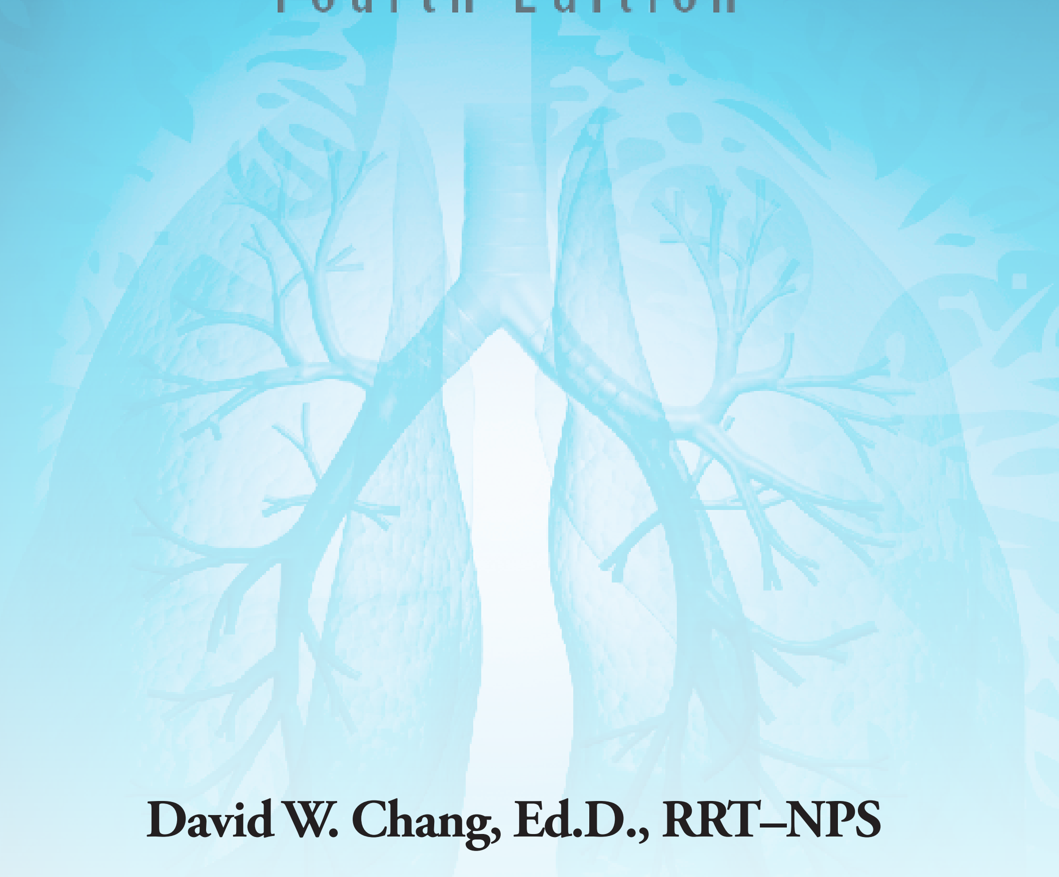
Clinical Application of MECHANICAL VENTILATION

Fourth Edition



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Dedicated with love
to my wife, Bonnie and our children,
Michelle, Jennifer, and Michael
for their support in my professional endeavors
and personal leisure activities

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Preface

Mechanical ventilation has been an integral part of critical care medicine. In its earlier years, ventilators were mainly used in the intensive care units and occasionally in the emergency departments for patient stabilization and intrahospital transport. In recent years, ventilators are used frequently in interhospital and intercontinental transport of critically ill patients. They are also used in mass casualty events, in both hyperbaric and hypobaric environments. Technology has evolved to a point where patients can manage the basic functions of their ventilators at home and even on a commercial aircraft.

Due to the inherited limitations of printed media, it would be impossible to provide adequate coverage on all topics, theories, procedures, and equipment related to mechanical ventilation. As a tradeoff, the primary focus of this mechanical ventilation textbook is to provide a basic but thorough presentation of those relevant topics that are pertinent to everyday clinical practice. Users of information technology and the Internet would agree that "more is not better." This book attempts to strike a balance between an adequate coverage in theory and a spectrum of needed clinical knowledge. The learners should find this book useful to develop a solid foundation in the theories of mechanical ventilation. With additional clinical experience, the learners should be able to integrate and apply the theories of mechanical ventilation in a clinical setting for better patient care.

In the fourth edition of *Clinical Application of Mechanical Ventilation*, new information and numerous references have been added. In some cases, older references are retained because their unique contribution has not been duplicated or cannot be found elsewhere. These classic references also allow learners and researchers to follow the path of progression in the knowledge and techniques of mechanical ventilation.

Overview of Textbook

In this fourth edition, the key terms are boldfaced within the text and the definitions are placed in the margin for quick reference. Essential information is also highlighted in the margin for quick reference. Learning objectives can be found in the beginning of Chapters 1 through 18.

Chapter 1 of the fourth edition reviews the normal pulmonary mechanics and the abnormal physiologic conditions leading to ventilatory failure. Chapter 2 provides a review of the effects of positive pressure ventilation on the major body systems and organs. Chapter 3 covers the components, terminology, and classification of mechanical ventilators. Chapter 4 describes up-to-date operating modes of mechanical ventilation. Chapter 5 reviews some special airways that are used to facilitate ventilation and oxygenation. Chapter 6 covers the application, management, and complications of endotracheal and tracheostomy tubes. Chapter 7 presents the clinical application of noninvasive positive pressure ventilation and the associated interfaces. Chapter 8 offers the common procedures for the initiation of mechanical ventilation. The indications, contraindications, initial ventilator settings, and alarm settings relating to mechanical ventilation are also discussed. Chapter 9 outlines the essential methods of patient monitoring to include imaging, fluid balance, blood gases, pulse oximetry, capnography, transcutaneous blood gases, and cerebral perfusion pressure. Chapter 10 covers the basics of invasive, less invasive and noninvasive hemodynamic monitoring. Chapter 11 gives a detailed discussion on ventilator waveform analysis and its applications. Chapter 12 presents the strategies to improve ventilation and oxygenation during mechanical ventilation. It also describes the basic strategies to manage ventilator alarms and abnormal physiologic conditions during mechanical ventilation. Chapter 13 reviews the basic pharmacotherapy for mechanical ventilation. The drugs discussed in this chapter include bronchodilators, neuromuscular blockers, central nervous agents, and other agents to facilitate patient comfort and patient-ventilator synchrony. Chapter 14 includes special procedures associated with mechanical ventilation—chest tube and drainage system, fiberoptic bronchoscopy, and transport of mechanically ventilated patients. Chapter 15 reviews some critical care issues in mechanical ventilation—acute lung injury, acute respiratory distress syndrome, ventilator-associated pneumonia, hypoxic-ischemic encephalopathy, and traumatic brain injury. Chapter 16 includes the criteria, procedure, and protocol for weaning from mechanical ventilation. Weaning failure and terminal weaning are also discussed. Chapter 17 covers a wide spectrum of neonatal mechanical ventilation to include high-frequency oscillatory ventilation and extracorporeal membrane oxygenation. In Chapter 18, mechanical ventilation in nontraditional settings is discussed. These settings include the use of a ventilator at home, in a mass casualty situation, in hyperbaric and hypobaric environments, as well as traveling with a mechanical ventilator on commercial aircraft. Chapter 19 has sixteen case studies related to mechanical ventilation.

New to This Edition

The fourth edition of *Clinical Application of Mechanical Ventilation* has two new chapters. Chapter 15 covers five critical care issues in mechanical ventilation that are commonly encountered by critical care providers. They are acute lung injury, acute respiratory distress syndrome, ventilator-associated pneumonia, hypoxic-ischemic encephalopathy, and traumatic brain injury. A recruitment maneuver to determine optimal PEEP is also included in Chapter 15. In Chapter 18, mechanical ventilation in nontraditional settings is discussed. These settings include the use of a ventilator at home, in a mass casualty situation, in hyperbaric and hypobaric environments, and on commercial aircraft. This new edition also provides much updated information. For example, modes of ventilation are updated in Chapter 4 to reflect current practice. Special visualization devices for intubation are added in Chapter 6. Less invasive and noninvasive hemodynamic monitoring techniques are added in Chapter 10. Weaning in progress and weaning protocols are updated in Chapter 16. In Chapter 19, a new case study covers the medical and ethical aspects of terminal weaning. The Appendices are updated to provide more useful reference information for the use and management of mechanical ventilation.

Ancillary Package

The complete supplement package for *Clinical Application of Mechanical Ventilation, fourth edition* was developed to achieve two goals:

1. To assist students in the learning and applying the information presented in the test.
2. To assist instructors in planning and implementing their courses in the most efficient manner and provide exceptional resources to enhance their students' experience.

Instructor Companion Website

ISBN 13: 978-1-111-53968-9

Spend less time planning and more time teaching with Delmar Cengage Learning's Instructor Resources to Accompany *Clinical Application of Mechanical Ventilation, fourth edition*. The Instructor Companion Website can be accessed by going to www.cengage.com/login to create a unique user log-in. The password-protected Instructor Resources include the following:

Instructor's Manual

An electronic instructor's manual provides instructors with invaluable tools for preparing for class lectures and examinations. The instructor's manual consists of three sections. The first section is a collection of potential test bank questions for each chapter, followed the second section that houses the answers for quick and easy assessment. The third section of the instructor's manual provides the answers to the workbook questions and exercises.

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An electronic testbank makes and generates tests and quizzes in an instant. With a variety of question types, including short answer, multiple choice, true or false, and matching exercises, creating challenging exams will be no barrier in your classroom. This testbank includes a rich bank of questions that test students on retention and application of what they've learned in the course. Answers are provided for all questions so instructors can focus on teaching, not grading.

Student Workbook

ISBN 13: 978-1-111-53967-2

The Student Workbook to accompany the fourth edition of Clinical Application of Mechanical Ventilation is a powerful learning aid for students and will enhance their comprehension and ability to apply what they have learned. Each workbook chapter follows the core textbook and supplies students with a variety of challenging exercises and quizzes to complete. This Workbook is a great asset to students and instructors alike to support active participation and engage the learning process.

Features of the Fourth Edition

The fourth edition includes many tried and true features that will enhance the learning experience and make this textbook a valuable asset in your education.

The addition of **Learning Objectives** listed at the beginning of each chapter outlines expected outcomes and is a great assessment tool after you've read the chapter. Another new feature is **Additional Resources,** which lists several assets in various media types that you can use to further your understanding of the chapter topics. Other features that offer guided study are a **Key Terms** list for each chapter and corresponding margin definitions for quick and easy reference. **Margin Notes** can be found throughout the chapters and succinctly present critical information for each chapter. Chapter **tables** and **figures** are improved with a brand new design and a second color to add prominence and draw attention to the information contained therein. Rounding out the important features of the fourth edition are the **Self-Assessment Questions** found at the end of each chapter that challenge you to apply the knowledge you've acquired throughout the chapter. **Answers** to the questions are included in each chapter for quick assessment to identify areas of weakness, and where further study is needed.

As in the past three editions, the goal of the fourth edition of *Clinical Application of Mechanical Ventilation* is to provide the students a textbook they will enjoy reading and using at school and at home. It is also my goal to make this textbook a quick reference source for respiratory care practitioners and other critical care providers.

—David W. Chang

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I would also like to recognize my colleagues who reviewed the contents of this edition for completeness and accuracy. Their help is very much appreciated throughout the development of this manuscript. They provided corrections, suggestions, and useful comments. The fourth edition of *Clinical Application of Mechanical Ventilation* should continue to be a useful textbook for students and a helpful reference source for critical care providers. The reviewers are:

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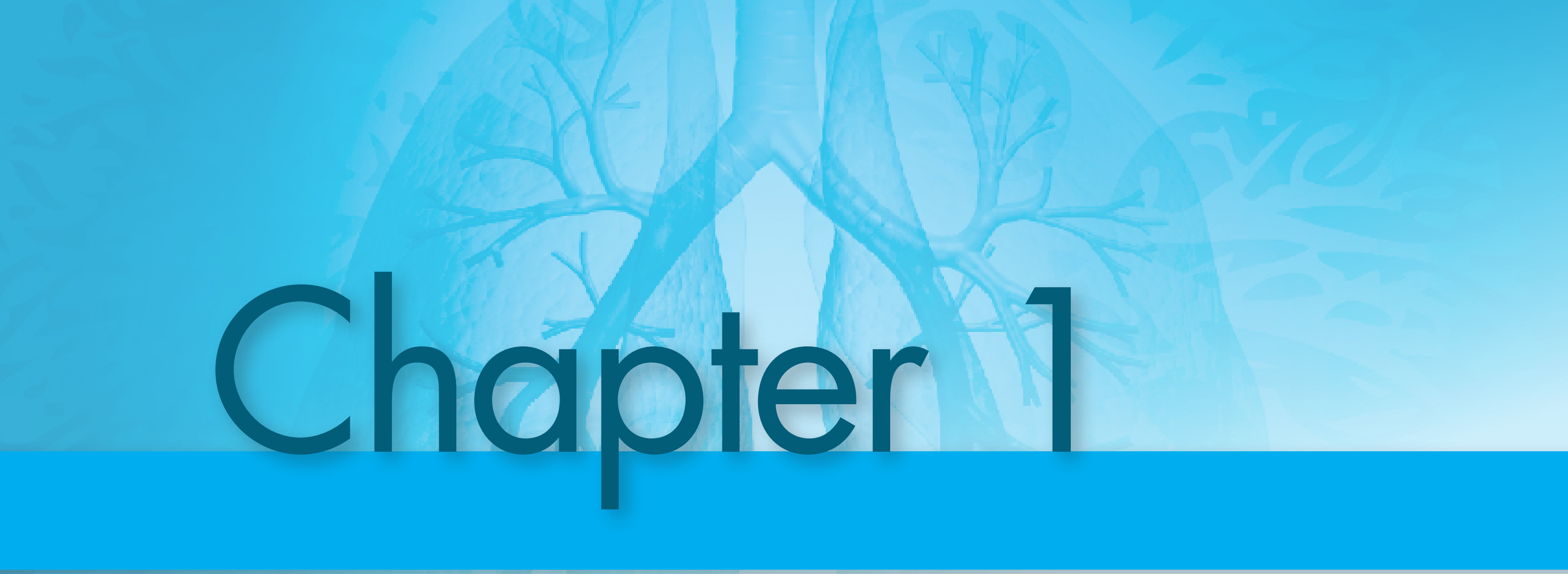

Chapter 1

Principles of Mechanical Ventilation

David W. Chang

Outline

- Introduction
- Airway Resistance
  - *Factors Affecting Airway Resistance*
  - *Airway Resistance and the Work of Breathing (ΔP)*
  - *Effects on Ventilation and Oxygenation*
  - *Airflow Resistance*
- Lung Compliance
  - *Compliance Measurement*
  - *Static and Dynamic Compliance*
  - *Compliance and the Work of Breathing*
  - *Effects on Ventilation and Oxygenation*
- Deadspace Ventilation
  - *Anatomic Deadspace*
  - *Alveolar Deadspace*
  - *Physiologic Deadspace*
- Ventilatory Failure
  - *Hypoventilation*
  - *Ventilation/Perfusion (V/Q) Mismatch*
  - *Intrapulmonary Shunting*
  - *Diffusion Defect*
- Oxygenation Failure
  - *Hypoxemia and Hypoxia*
- Clinical Conditions Leading to Mechanical Ventilation
  - *Depressed Respiratory Drive*
  - *Excessive Ventilatory Workload*
  - *Failure of Ventilatory Pump*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

airway resistance
alveolar deadspace
alveolar volume
anatomic deadspace
deadspace ventilation
diffusion defect

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hypoventilation
hypoxic hypoxia
intrapulmonary shunting
lung compliance
oxygenation failure
peak inspiratory pressure
physiologic deadspace
plateau pressure
refractory hypoxemia
ventilatory failure
V/Q mismatch

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Use required variables and calculate airway resistance, compliance, and deadspace ventilation.
- Describe the relationship among the three variables in airway resistance, compliance, and deadspace ventilation.
- Describe the clinical application of static and dynamic compliance.
- Explain the changes in airway resistance, lung compliance, and deadspace ventilation that contribute to the increased work of breathing and ventilatory failure.
- Describe the process of four clinical conditions that lead to ventilatory failure.
- Identify the presence of hypoxemia and signs of hypoxia.
- Describe three primary clinical conditions that lead to mechanical ventilation.

INTRODUCTION

Mechanical ventilation is a useful modality for patients who are unable to sustain the level of ventilation necessary to maintain the gas exchange functions (oxygenation and carbon dioxide elimination). Indications for mechanical ventilation vary greatly among patients. Mechanical ventilation may be indicated in conditions due to physiologic changes (e.g., deterioration of lung parenchyma), disease states (e.g., respiratory distress syndrome), medical/surgical procedures (e.g., postanesthesia recovery), and many other causes (e.g., head trauma, drug overdose) leading to ventilatory failure or oxygenation failure.

Use of mechanical ventilation also varies greatly from short term to long term and from acute care in the hospital to extended care at home. One of the most frequent uses of mechanical ventilation is for the management of postoperative patients recovering from anesthesia and medications. Other indications for mechanical ventilation in adults include apnea and impending respiratory arrest, acute exacerbation of COPD, acute severe asthma, neuromuscular disease, acute hypoxemic respiratory failure, heart failure and cardiogenic shock, acute brain injury, and flail chest (Pierson, 2002).

Regardless of the diagnosis or disease state, patients who require mechanical ventilation generally have developed ventilatory failure, oxygenation failure, or both. Specifically, when a patient fails to ventilate or oxygenate adequately, the problem may be caused by one of six major pathophysiological factors: (1) increased airway resistance, (2) changes in lung compliance, (3) hypoventilation, (4) V/Q mismatch, (5) intrapulmonary shunting, or (6) diffusion defect.

AIRWAY RESISTANCE

airway resistance: The degree of airflow obstruction in the airways.

Airway resistance is defined as airflow obstruction in the airways. In mechanical ventilation, the degree of airway resistance is primarily affected by the length, size, and patency of the airway, endotracheal tube, and ventilator circuit.

Factors Affecting Airway Resistance

Airway resistance causes obstruction of airflow in the airways. It is increased when the patency or diameter of the airways is reduced. Obstruction of airflow may be caused by: (1) changes inside the airway (e.g., retained secretions), (2) changes in the wall of the airway (e.g., neoplasm of the bronchial muscle structure), or (3) changes outside the airway (e.g., tumors surrounding and compressing the airway) (West, 2007). When one of these conditions occurs, the radius of the airway decreases and airway resistance increases. According to the simplified form of Poiseuille's Law, the driving pressure (ΔP) to maintain the same airflow ($\dot{V}$) must increase by a factor of 16-fold when the radius (r) of the airway is reduced by only half of its original size.

$$\text{Simplified form of Poiseuille's Law: } \Delta P = \frac{\dot{V}}{r^4}$$

✔ Based on Poiseuille's Law, the work of breathing increases by a factor of 16-fold when the radius (r) of the airway is reduced by half its original size.

One of the most common causes of increased airway resistance is chronic obstructive pulmonary disease (COPD). This type of lung disease includes emphysema, chronic bronchitis, chronic asthma, and bronchiectasis. Mechanical conditions that may increase airway resistance include postintubation obstruction and foreign body aspiration. Infectious processes include laryngotracheobronchitis (croup), epiglottitis, and bronchiolitis. Table 1-1 lists three categories of clinical conditions that increase airway resistance.

✔ Airway resistance varies directly with the length and inversely with the diameter of the airway or ET tube.

Normal airway resistance in healthy adults is between 0.5 and 2.5 cm H_2O/L/sec (Wilkins, 2009). It is higher in intubated patients due to the smaller diameter of the endotracheal (ET) tube. Airway resistance varies directly with the length and inversely with the diameter of the airway or ET tube. In the clinical setting, the ET tube may be shortened for ease of airway management, reduction of mechanical deadspace, and reduction of airway resistance. However, the major contributor to increased airway resistance is the internal diameter of the ET tube. Therefore, during intubation, the largest appropriate size ET tube must be used so that the airway resistance contributed by the ET tube may be minimized. Once the ET tube is in place,

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TABLE 1-1 Clinical Conditions That Increase Airway Resistance

Type	Clinical Conditions
COPD	Emphysema Chronic bronchitis Asthma Bronchiectasis
Mechanical obstruction	Postintubation obstruction Foreign body aspiration Endotracheal tube Condensation in ventilator circuit
Infection	Laryngotracheobronchitis (croup) Epiglottitis Bronchiolitis

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its patency must be maintained, as secretions inside the ET tube greatly increase airway resistance.

Besides the ET tube, the ventilator circuit may also impose mechanical resistance to airflow and contribute to total airway resistance. This is particularly important when there is a significant amount of water in the ventilator circuit due to condensation. Chapter 4 describes the use of pressure support ventilation (PSV) to compensate for the effects of airflow resistance and to augment spontaneous tidal volume during mechanical ventilation.

Airway Resistance and the Work of Breathing (ΔP)

Airway resistance is calculated by $\frac{\text{Pressure Change}}{\text{Flow}}$

$$\text{Raw} = \frac{\Delta \text{P}}{\dot{\text{V}}}$$

Raw = airway resistance

ΔP = pressure change (Peak Inspiratory Pressure − Plateau Pressure)

$\dot{\text{V}}$ = Flow

↑ Airway resistance = ↑ Work of breathing.

The pressure change (ΔP) in the equation reflects the work of breathing imposed on the patient. Since airway resistance is directly related to pressure change (the work of breathing), an increase in airway resistance means the patient must exert more energy for ventilation. In a clinical setting, relief of airflow obstruction is an effective way to reduce the work of breathing (Blanch et al., 2005; Myers, 2006).

If pressure change (work of breathing) in the equation above is held constant, an increase in airway resistance will cause a decrease in flow and subsequently a decrease

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in minute ventilation. This is because airway resistance and flow in the equation are *inversely* related. In a clinical setting, **hypoventilation** may result if the patient is unable to overcome the airway resistance by increasing the work of breathing.

hypoventilation: Inadequacy of ventilation to remove CO_2. The arterial PCO_2 is elevated in conditions of hypoventilation.

As a result of chronic air trapping, patients with chronic airway obstruction may develop highly compliant lung parenchyma. These patients use a breathing pattern that is deeper but slower. On the other hand, patients with restrictive lung disease (low compliance) breathe more shallowly but faster, since airflow resistance is not the primary disturbance in these patients.

Effects on Ventilation and Oxygenation

The work of breathing imposed on a patient is increased when airway resistance is high. This creates a detrimental effect on the patient's ventilatory and oxygenation status. If an abnormally high airway resistance is sustained over a long time, fatigue of the respiratory muscles may occur, leading to ventilatory and oxygenation failure (Rochester, 1993). **Ventilatory failure** occurs when the patient's minute ventilation cannot keep up with CO_2 production. **Oxygenation failure** usually follows when the cardiopulmonary system cannot provide adequate oxygen needed for metabolism.

ventilatory failure: Failure of the respiratory system to remove CO_2 from the body resulting in an abnormally high $PaCO_2$.

oxygenation failure: Failure of the heart and lungs to provide adequate oxygen for metabolic needs.

✓ An increased bowing of the P-V loop suggests an overall increase in airflow resistance.

Airflow Resistance

The airflow resistance of a patient-ventilator system may be monitored using the pressure-volume (P-V) loop display on a ventilator waveform display (Waugh et al., 2007). An increased bowing of the P-V loop suggests an overall increase in airflow resistance (Figure 1-1). The increase in airflow resistance may be caused by excessive inspiratory flow or increased expiratory flow resistance.

✓ Excessive inspiratory flow → bowing of inspiratory limb. (A_1 to A_2)

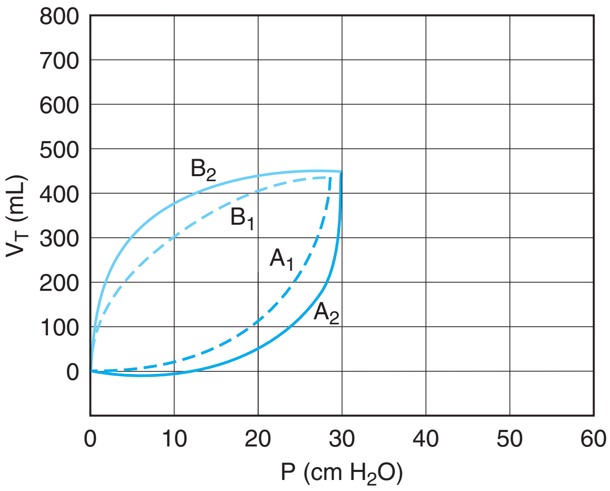

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FIGURE 1-1 Increased bowing (from dotted to solid lines) of the pressure–volume loop suggests an increase in airflow resistance. Bowing of inspiratory limb (from A_1 to A_2) may be caused by excessive inspiratory flow. Bowing of the expiratory limb (from B_1 to B_2) may be caused by an increase in expiratory flow resistance such as bronchospasm.

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✓ Increased expiratory flow resistance → bowing of expiratory limb. (B_1 to B_2)

When the inspiratory flow exceeds a patient's tidal volume and inspiratory time requirement, bowing of the inspiratory limb may result (line A_2). In situations where the expiratory airflow resistance is increased (e.g., bronchospasm), bowing of the expiratory limb (line B_2) may occur.

LUNG COMPLIANCE

lung compliance: The degree of lung expansion per unit pressure change.

Lung compliance is volume change (lung expansion) per unit pressure change (work of breathing), and it is calculated by $C = \Delta V/\Delta P$, where C = compliance, ΔV = volume change, and ΔP = pressure change. Most ventilators can measure and show the static and dynamic compliance values on the data or graphic display. A method to measure and calculate static and dynamic compliance is outlined in Table 1-2.

Compliance Measurement

refractory hypoxemia: A persistent low level of oxygen in blood that is not responsive to medium to high concentration of inspired oxygen. It is usually caused by intrapulmonary shunting.

plateau pressure: The pressure needed to maintain lung inflation in the absence of airflow.

peak inspiratory pressure: The pressure used to deliver the tidal volume by overcoming nonelastic (airways) and elastic (lung parenchyma) resistance.

Abnormally low or high lung compliance impairs the patient's ability to maintain efficient gas exchange. Low compliance typically makes lung expansion difficult. High compliance induces incomplete exhalation, air trapping, and reduced CO_2 elimination. These abnormalities are often contributing factors to the need for mechanical ventilation.

Low Compliance. Low compliance (high elastance) means that the volume change is small per unit pressure change. Under this condition, the lungs are *stiff* or *noncompliant*. The work of breathing is increased when the compliance is low. In many clinical situations (e.g., acute respiratory distress syndrome or ARDS), low lung compliance is associated with **refractory hypoxemia**.

TABLE 1-2 Method to Measure Static and Dynamic Compliance

(1) Obtain corrected expired tidal volume.

(2) Obtain **plateau pressure** by applying inspiratory hold or occluding the exhalation port at end-inspiration.

(3) Obtain **peak inspiratory pressure.**

(4) Obtain positive end-expiratory pressure (PEEP) level, if any.

$$\text{Static Compliance} = \frac{\text{Corrected Tidal Volume}}{(\text{Plateau Pressure} - \text{PEEP})}$$

$$\text{Dynamic Compliance} = \frac{\text{Corrected Tidal Volume}}{(\text{Peak Inspiratory Pressure} - \text{PEEP})}$$

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TABLE 1-3 Clinical Conditions That Decrease the Compliance

Type of Compliance	Clinical Conditions
Static compliance	ARDS Atelectasis Tension pneumothorax Obesity Retained secretions
Dynamic compliance	Bronchospasm Kinking of ET tube Airway obstruction

✓ ↓ Lung compliance = ↑ Work of breathing.

Low compliance measurements are usually related to conditions that reduce the patient's functional residual capacity. Patients with noncompliant lungs often have a restrictive lung defect, low lung volumes, and low minute ventilation. This condition may be compensated for by an increased frequency. Table 1-3 shows some examples that lead to a decreased compliance measurement.

✓ In extreme high compliance situations, exhalation is often incomplete due to reduced elastic recoil of the lungs.

High Compliance. High compliance means that the volume change is large per unit pressure change. In extreme high compliance situations, exhalation is often incomplete due to lack of elastic recoil by the lungs. Emphysema is an example of high compliance where the gas exchange process is impaired. This condition is due to chronic air trapping, destruction of lung tissues, and enlargement of terminal and respiratory bronchioles.

High compliance measurements are usually related to conditions that increase the patient's functional residual capacity and total lung capacity. Patients with extremely compliant lungs often develop airflow obstruction, incomplete exhalation, air trapping, and poor gas exchange.

Static and Dynamic Compliance

Assessment of compliance can be divided into static compliance and dynamic compliance measurements. The relationship and clinical significance of these measurements are discussed in the following sections.

✓ Static compliance reflects the elastic properties (elastic resistance) of the lung and chest wall.

Static Compliance. Static compliance is calculated by dividing the volume by the pressure (i.e., plateau pressure) measured when the flow is momentarily stopped. When airflow is absent, airway resistance becomes a non-factor. Static compliance reflects the elastic resistance of the lung and chest wall.

✓ Dynamic compliance reflects the airway resistance (nonelastic resistance) and the elastic properties of the lung and chest wall (elastic resistance).

Dynamic Compliance. Dynamic compliance is calculated by dividing the volume by the pressure (i.e., peak inspiratory pressure) measured when airflow is present. Since airflow is present, airway resistance becomes a factor in the measurement of dynamic compliance. Dynamic compliance therefore reflects the condition of airway

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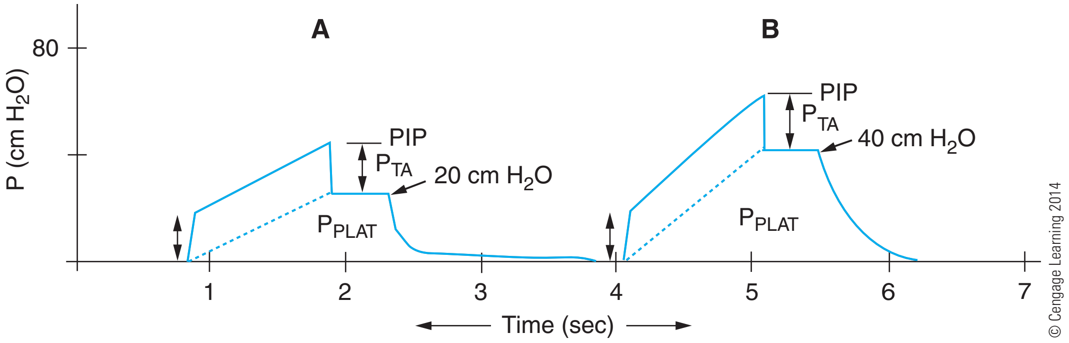

FIGURE 1-2 In conditions where the lung compliance is decreased (e.g., atelectasis), the plateau pressure (P_{PLAT}) and peak inspiratory pressure (PIP) are both increased (from A to B).

resistance (nonelastic resistance) as well as the elastic properties of the lung and chest wall (elastic resistance).

✔ Conditions causing changes in plateau pressure and static compliance invoke similar changes in peak inspiratory pressure and dynamic compliance.

✔ When the airflow resistance is increased (e.g., bronchospasm), the peak inspiratory pressure is increased while the plateau pressure stays unchanged.

Plateau and Peak Inspiratory Pressure. In general, conditions causing changes in plateau pressure and static compliance invoke similar changes in peak inspiratory pressure and dynamic compliance. For example, atelectasis causes an increase of plateau and peak inspiratory pressures (Figure 1-2, A to B). Since the plateau and peak inspiratory pressures are increased, the calculated static and dynamic compliance measurements are decreased. When atelectasis is resolved, the plateau and peak inspiratory pressures return to normal (Figure 1-2, B to A).

In conditions where the airflow resistance is increased (e.g., bronchospasm), the peak inspiratory pressure is increased while the plateau pressure stays unchanged (Figure 1-3, A to B). Since the peak inspiratory pressure is increased, the dynamic compliance is decreased. The static compliance stays the same because there is no change in the plateau pressure. When bronchospasm is resolved, the peak inspiratory pressure and dynamic compliance measurements return to their previous states. (Figure 1-3, B to A).

Pressure-Volume Loop. Since compliance is determined by $\Delta V/\Delta P$, the P-V loop is essentially a "compliance loop," and it provides useful information on the characteristics of a patient's compliance. Figure 1-4 shows a P-V loop during a mandatory

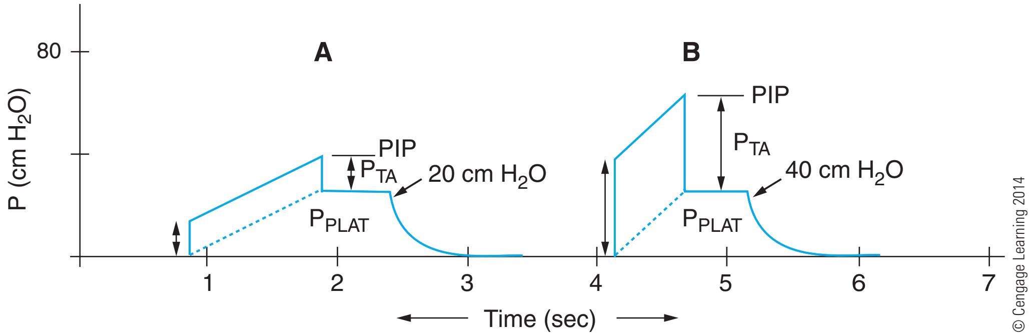

FIGURE 1-3 In conditions where the airflow resistance is increased (e.g., bronchospasm), the peak inspiratory pressure (PIP) is increased while the plateau pressure (P_{PLAT}) stays unchanged (from A to B).

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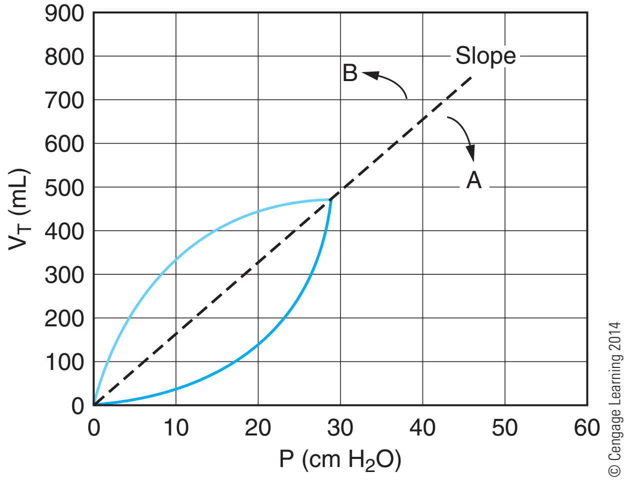

FIGURE 1-4 Shifting of the P-V slope toward the pressure axis (A) indicates a decrease in compliance. Shifting of the P-V slope toward the volume axis (B) indicates an increase in compliance.

✓ A shift of the slope toward the pressure axis indicates a decrease in compliance.

✓ A shift of the slope toward the volume axis indicates an increase in compliance.

breath. A slope is drawn from the beginning point dividing the inspiratory limb and the expiratory limb. A shift of the slope toward the pressure axis indicates a decrease in compliance. A shift of the slope toward the volume axis indicates an increase in compliance (Waugh et al., 2007).

In another P-V loop (Figure 1-5), a shift of the slope and the entire P-V loop toward the pressure axis shows an increase in pressure (↑ ΔP) required to deliver the same volume (ΔV). This condition shows a decrease in compliance (↓C) (Waugh et al., 2007).

Compliance measurements should be made so that a trend can be established. Interpretation is of little value with a single compliance measurement. It is also essential not to compare static compliance with dynamic compliance measurements as this can cause erroneous and meaningless interpretations.

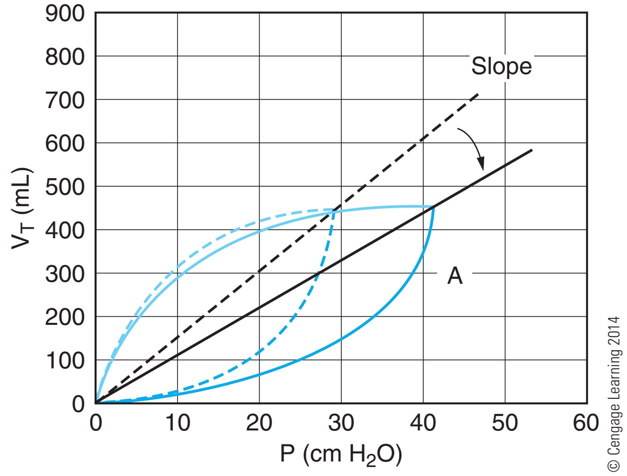

FIGURE 1-5 Shifting of the P-V slope and loop toward the pressure axis shows a higher pressure is required to deliver the same volume (decrease in compliance).

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Clinical Ranges. For critically ill patients, the dynamic compliance is between 30 and 40 mL/cm H_2O and static compliance is between 40 and 60 mL/cm H_2O (Barnes et al., 1994). It is lower in intubated patients, depending on the internal diameter of the ET tube. Refer to Table 1-2 for the method to measure compliance. The equations for static compliance (C_{ST}) and dynamic compliance (C_{DYN}) are:

✔ See Appendix 1 for example.

$$C_{ST} = \frac{\text{Corrected Tidal Volume}}{(\text{Plateau Pressure} - \text{PEEP})}$$

$$C_{DYN} = \frac{\text{Corrected Tidal Volume}}{(\text{Peak Inspiratory Pressure} - \text{PEEP})}$$

Compliance and the Work of Breathing

Since compliance is inversely related to pressure change (work of breathing), a decrease in compliance means an increase in the work of breathing. In a clinical setting, acute respiratory distress syndrome (ARDS) and atelectasis are two causes of increased work of breathing.

If the plateau pressure and peak inspiratory pressure (work of breathing) in the previous equations are held unchanged, a decrease in compliance will cause a decrease in volume. This is because compliance and volume change in the equations are directly related. In a clinical setting, hypoventilation usually results when a patient is unable to compensate for the decrease in compliance by increasing and maintaining a higher level of work of breathing. In low compliance situations, such as ARDS, pulmonary fibrosis, and kyphoscoliosis, the decrease in minute ventilation is characterized by decreased tidal volumes and increased frequencies—a sign of volume restriction.

Effects on Ventilation and Oxygenation

Abnormal compliance impairs the gas exchange mechanism. When an abnormally low or high compliance is uncorrected and prolonged, muscle fatigue may occur and lead to the development of ventilatory and oxygenation failure (Rochester, 1993). Ventilatory failure develops when the patient's minute ventilation cannot keep up with the CO_2 production. Oxygenation failure usually follows when the cardiopulmonary system cannot supply the oxygen needed for metabolism and prolonged increase in the work of breathing.

DEADSPACE VENTILATION

deadspace ventilation: Ventilation in excess of perfusion; wasted ventilation.

Deadspace ventilation is defined as wasted ventilation or a condition in which ventilation is in excess of perfusion. There are three types of deadspace: anatomic, alveolar, and physiologic.

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Anatomic Deadspace

anatomic deadspace: The volume occupying the conducting airways that does not take part in gas exchange (estimated to be 1 mL/lb ideal body weight).

✓ Decrease in tidal volume causes a relatively higher anatomic deadspace to tidal volume percent.

Normally, the conducting airways contribute to about 30% of deadspace ventilation. For a tidal volume of 500 mL, about 150 mL of this volume is wasted since it does not take part in gas exchange. This volume in the conducting airways is called **anatomic deadspace** and it can be estimated to be 1 mL/lb of ideal body weight (Shapiro et al., 1991).

Decrease in tidal volume causes a *relatively* higher anatomic deadspace to tidal volume percent. For example, if the tidal volume was decreased from 500 to 300 mL, the deadspace to tidal volume percent would increase from 30% (150/500) to 50% (150/300) See equations below for comparison:

$$\frac{150}{500} = 0.3 \text{ or } 30\%$$

$$\frac{150}{300} = 0.5 \text{ or } 50\%$$

Alveolar Deadspace

alveolar deadspace: The normal lung volume that has become unable to take part in gas exchange because of reduction or lack of pulmonary perfusion (e.g., pulmonary embolism).

In addition to anatomic deadspace, **alveolar deadspace** may occur in some clinical conditions. Alveolar deadspace contributes to wasted ventilation, and it occurs when the ventilated alveoli are not adequately perfused by pulmonary circulation. Pulmonary perfusion may be absent or low because of decreased cardiac output (e.g., congestive heart failure, blood loss), or due to obstruction of the pulmonary blood vessels (e.g., pulmonary vasoconstriction, pulmonary embolism) (Shapiro et al., 1991). Figure 1-6 shows the relationship between ventilation and perfusion during alveolar deadspace ventilation.

Physiologic Deadspace

physiologic deadspace: Sum of anatomic and alveolar deadspace. Under normal conditions, it is about the same as anatomic deadspace.

Physiologic deadspace is the sum of anatomic and alveolar deadspace volumes. Under normal conditions, the physiologic deadspace approximates the anatomic deadspace. In diseased conditions where alveolar deadspace ventilation is increased, physiologic deadspace becomes higher than anatomic deadspace. Table 1-4 shows some clinical conditions that increase physiologic (anatomic and alveolar) deadspace.

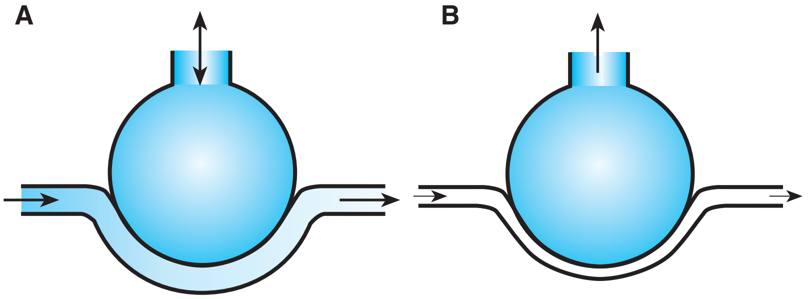

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FIGURE 1-6 (A) Normal ventilation/perfusion relationship; (B) Alveolar deadspace ventilation occurs when the ventilated alveoli are not adequately perfused by pulmonary circulation (i.e., ventilation in excess of perfusion). Examples of deadspace ventilation include decrease in cardiac output and obstruction of pulmonary blood vessels.

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TABLE 1-4 Clinical Conditions That Increase Physiologic Deadspace

Type of Change	Clinical Conditions
↓ Tidal volume	Relative increase in V_D/V_T (drug overdose, neuromuscular disease)
↑ Alveolar deadspace	Decreased cardiac output (congestive heart failure, blood loss) Obstruction of pulmonary blood vessels (pulmonary vasoconstriction, pulmonary embolism)

Physiologic deadspace to tidal volume ratio (VD/VT) can be calculated as follows:

$$\frac{V_D}{V_T} = \frac{(PaCO_2 - P_E\text{-}CO_2)}{PaCO_2}$$

✓ See Appendix 1 for example.

$PaCO_2$ is arterial carbon dioxide tension and $P_E\text{-}CO_2$ is PCO_2 of a mixed expired gas sample. These two samples are collected simultaneously. In patients on mechanical ventilation, V_D/V_T of less than 60% is considered acceptable and this value suggests adequate ventilatory function upon weaning from mechanical ventilation (Shapiro et al., 1991).

Severe and prolonged deadspace ventilation causes inefficient ventilation, muscle fatigue, and ventilatory and oxygenation failure.

VENTILATORY FAILURE

Ventilatory failure is the inability of the pulmonary system to maintain proper removal of carbon dioxide. Hypercapnia (increase in $PaCO_2$) is the key feature of ventilatory failure. When carbon dioxide production exceeds its removal, respiratory acidosis results. Hypoxemia can be the secondary complication of ventilatory failure. In general, hypoxemia due to hypoventilation responds well to ventilation and low concentration of supplemental oxygen. Without supplemental oxygen, the degree of hypoxemia corresponds to the severity of ventilatory failure.

V/Q mismatch: An abnormal distribution of ventilation and pulmonary blood flow. High V/Q is related to deadspace ventilation, whereas low V/Q is associated with intrapulmonary shunting.

intrapulmonary shunting: Pulmonary blood flow in excess of ventilation; wasted perfusion (e.g., atelectasis).

diffusion defect: Pathologic condition leading to impaired gas exchange through the alveolar-capillary membrane (e.g., interstitial or pulmonary edema).

Table 1-5 lists five mechanisms leading to the development of ventilatory failure. They are (1) hypoventilation, (2) persistent ventilation/perfusion (**V/Q**) **mismatch**, (3) persistent **intrapulmonary shunting**, (4) persistent **diffusion defect**, and (5) persistent reduction of inspired oxygen tension (P_IO_2) (Greene et al., 1994).

Hypoventilation

Hypoventilation can be caused by depression of the central nervous system, neuromuscular disorders, airway obstruction, and other conditions. In a clinical setting, hypoventilation is characterized by a reduction of alveolar ventilation (V_A) and an increase of arterial carbon dioxide tension ($PaCO_2$).

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TABLE 1-5 Development of Ventilatory Failure

Mechanism	Clinical Finding
Hypoventilation	$PaCO_2$ greater than 45 mm Hg (>50 mm Hg for patients with COPD)
Persistent V/Q mismatch	Hypoxemia that responds well to oxygen therapy
Persistent intrapulmonary shunt	Q_{SP}/Q_T greater than 20% (>30% in critical shunt)
Persistent diffusion defect	Gas diffusion rate less than 75% of predicted normal
Persistent reduction of P_IO_2	Low barometric pressure as in high altitude

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alveolar volume: The portion of tidal volume that takes part in gas exchange.

Alveolar Volume. **Alveolar volume** (V_A) is the difference between tidal volume (V_T) and deadspace volume (V_D):

$$V_A = V_T - V_D$$

The equation shows that alveolar volume can be increased by raising the tidal volume or by reducing the deadspace volume. In mechanical ventilation, a reduction in alveolar volume occurs when the tidal volume delivered to the patient is decreased or the deadspace volume is increased.

Hypoventilation caused by a reduction in tidal volume can be corrected by increasing the tidal volume (in volume-controlled ventilation) or the peak inspiratory pressure (in pressure-controlled ventilation) on the ventilator. Unlike tidal volume, deadspace volume is difficult to change because anatomic deadspace stays rather constant and physiologic deadspace is due to decreased perfusion. Alveolar hypoventilation, due to a decrease in perfusion, requires improvement of the pulmonary blood flow.

✓ $\dot{V}_A = (V_T - V_D) \times f$ (See Appendix 1 for example).

Minute Alveolar Ventilation ($\dot{V}_A$). Minute alveolar ventilation ($\dot{V}_A$) is a function of the tidal volume, deadspace volume, and respiratory frequency per minute. Hypoventilation can result when the frequency is too slow or absent (apnea). Hypoventilation due to a reduction in spontaneous frequency can be compensated by increasing the frequency (assist or SIMV) on the ventilator.

✓ Hypoventilation is characterized by an increase of $PaCO_2$.

A patient's ventilatory status can best be monitored by the $PaCO_2$ measurement. The equation below shows the inverse relationship between $\dot{V}_A$ and $PaCO_2$. When the minute alveolar ventilation is low (hypoventilation), an elevated $PaCO_2$ is the typical finding in blood gas analysis.

$$\dot{V}_A = \frac{\dot{V}CO_2}{PaCO_2}$$

Ventilation/Perfusion (V/Q) Mismatch

The ventilation/perfusion (V/Q) ratio is the numeric representation (in percent) of ventilation in relation to the amount of pulmonary blood flow (perfusion). Since blood flow is gravity-dependent, the V/Q ratio ranges from about 0.4 in the

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lower lung zone (more perfusion) to 3.0 in the upper lung zone (less perfusion) (West, 2008).

In disease conditions, pulmonary embolism decreases pulmonary perfusion and leads to a high V/Q. Airway obstruction is one example that leads to decrease in ventilation and low V/Q.

V/Q mismatch is responsible for the development of hypoxemia. With sufficient pulmonary reserve, a patient can usually compensate for the hypoxemic condition by hyperventilation. Hypoxemia caused by uncomplicated V/Q mismatch is readily reversible by oxygen therapy.

In mechanical ventilation, hypoxemia caused by V/Q mismatch can be compensated by increasing the frequency, tidal volume, or F_IO_2 on the ventilator (Shapiro et al., 1991).

Intrapulmonary Shunting

Shunted pulmonary blood flow is not useful in gas exchange.

Intrapulmonary shunting causes refractory hypoxemia.

In contrast with deadspace ventilation (ventilation in excess of perfusion), shunting refers to perfusion in excess of ventilation ("wasted" perfusion) (Figure 1-7). Shunted pulmonary blood flow is ineffective in gas exchange because it does not come in contact with ventilated and oxygenated alveoli. Intrapulmonary shunting causes refractory hypoxemia.

In healthy individuals, the physiologic shunt approximates the anatomic shunt and it is less than 5%. For noncritically ill patients, the normal physiologic shunt is less than 10%. In other disease states, the physiologic shunt may be greater than 30% (Shapiro et al., 1994). See Table 1-6 for interpretation of shunt percent in hospitalized patients.

The shunt percent can be calculated or estimated by many methods, ranging from simple (less accurate) to complex (more accurate). The clinical use of two common calculations are discussed here: an estimated shunt equation and a classic shunt equation.

The estimated physiologic shunt equation requires only an arterial blood sample.

Estimated Physiologic Shunt Equation. The estimated physiologic shunt equation requires only an arterial blood sample. It does not require a mixed venous blood sample from the pulmonary artery, and therefore it is noninvasive and rather simple to compute. This estimated method is more meaningful when serial measurements are used to establish a trend. Two forms of this equation are possible:

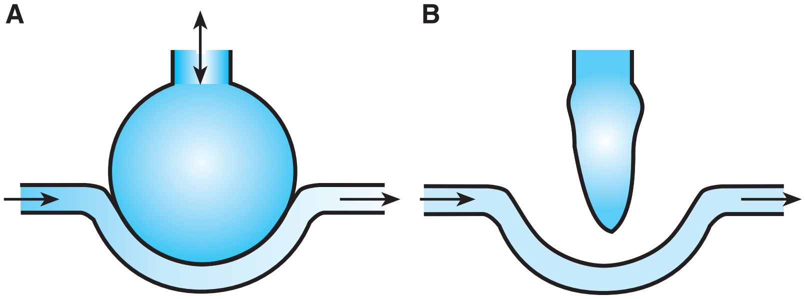

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FIGURE 1-7 (A) Normal ventilation/perfusion relationship; (B) Intra-pulmonary shunting occurs when the perfused alveoli are not adequately ventilated (i.e., perfusion in excess of ventilation). Atelectasis is an example that leads to intrapulmonary shunting.

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TABLE 1-6 Interpretation of Shunt Percent

Physiologic Shunt	Interpretation
$<10\%$	Normal
10% to 20%	Mild shunt
20% to 30%	Significant shunt
$>30\%$	Critical and severe shunt

one for noncritical patients (e.g., spontaneous breathing, moderate level of F_IO_2, moderate level of continuous positive airway pressure) and one for critically ill patients (e.g., mechanical ventilation, high F_IO_2, high level of positive end-expiratory pressure).

✔ See Appendix 1 for example.

For noncritical patient:

$$\text{Estimated } \frac{Q_{SP}}{Q_T} = \frac{(CcO_2 - CaO_2)}{[5 + (CcO_2 - CaO_2)]}$$

For critical patient:

$$\text{Estimated } \frac{Q_{SP}}{Q_T} = \frac{(CcO_2 - CaO_2)}{[3.5 + (CcO_2 - CaO_2)]}$$

✔ The classic physiologic shunt equation requires arterial and mixed venous blood samples.

Classic Physiologic Shunt Equation. The classic physiologic shunt equation requires an arterial blood sample and a mixed venous blood sample from the pulmonary artery. It is the most accurate among all shunt equations.

$$\text{Classic } \frac{Q_{SP}}{Q_T} = \frac{(CcO_2 - CaO_2)}{(CcO_2 - C\dot{v}O_2)}$$

✔ See Appendix 1 for example.

When the shunt percent is too high, oxygenation becomes an extremely difficult task for the cardiopulmonary system to support. Over time, the respiratory muscles fatigue, resulting in ventilatory failure. This is usually followed by oxygenation failure if ventilatory interventions are unsuccessful (Rochester, 1993).

Since intrapulmonary shunting is a result of lack of adequate ventilation due to collapsed or fluid-filled lung units, positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) may be used to open up and ventilate these lung units. Chapter 4 describes the use of PEEP or CPAP in the management of intrapulmonary shunting during mechanical ventilation (PEEP) or spontaneous breathing (CPAP).

Diffusion Defect

Diffusion of gases (including oxygen and carbon dioxide) across the alveolar-capillary (A-C) membrane is mainly dependent on the gas pressure gradients. Oxygen diffuses from the alveoli ($P_AO_2 = 109$ mm Hg) to the pulmonary arterial capillaries ($P\dot{v}O_2 = 40$ mm Hg) with a pressure gradient of 69 mm Hg. Carbon

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TABLE 1-7 Causes of Decreased Diffusion Rate

Type of Diffusion Problem	Clinical Conditions
Decrease in $P(A\text{-}a)O_2$ gradient	High altitude Fire combustion
Thickening of A-C membrane	Pulmonary edema Retained secretions
Decreased surface area of A-C membrane	Emphysema Pulmonary fibrosis
Insufficient time for diffusion	Tachycardia

dioxide diffuses from the pulmonary arterial capillaries ($P\dot{v}CO_2 = 46$ mm Hg) to the alveoli ($P_ACO_2 = 40$ mm Hg) with a net pressure gradient of only 6 mm Hg. This is possible because the gas diffusion coefficient for carbon dioxide is 19 times greater than that for oxygen.

Diffusion of oxygen is greatly impaired when the inspired oxygen tension (P_IO_2) is reduced. The inspired oxygen tension is directly related to the barometric pressure. At high altitude where the barometric pressure is low, the inspired oxygen tension is also low. This leads to a condition known as **hypoxic hypoxia**.

hypoxic hypoxia: Lack of oxygen in the organs and tissues due to a reduction in inspired oxygen tension.

The P_IO_2 is also reduced in a burning enclosure as combustion consumes oxygen in the air. Patients who suffer from smoke inhalation are at risk for developing hypoxic hypoxia. In addition to the reduced oxygen concentration and tension, the lung functions are impaired by the presence of carbon monoxide, toxic gases, and inert particles found in a burning environment (Wilkins, 1998).

In addition to the pressure gradient and diffusion coefficient, the gas diffusion rate is also affected by the thickness of the A-C membrane, the surface area of the A-C membrane, and the time available for diffusion to take place. Factors that can decrease the diffusion rate are shown in Table 1-7.

Conditions in Table 1-7 induce poor or inadequate gas diffusion and can severely hinder the oxygenation process. Hypoxemia and hypoxia are usually the end results. Severe hypoxemia and hypoxia may lead to hypoxic pulmonary vasoconstriction, pulmonary hypertension, and cor pulmonale.

OXYGENATION FAILURE

Oxygenation failure is defined as severe hypoxemia that does not respond to moderate to high levels of supplemental oxygen. It may be caused by hypoventilation, ventilation/perfusion mismatch, or intrapulmonary shunting. Regardless of the etiology of oxygenation failure, mechanical ventilation may be needed to minimize the work of breathing and provide oxygenation support.

Hypoxemia and Hypoxia

Hypoxemia is present when the oxygen level (e.g., PO_2, SaO_2) is decreased in arterial blood. The presence of hypoxia (↓PO_2 in organs and tissues) may not be always apparent. Hypoxemia reflects the likelihood of hypoxia, but hypoxia can occur in the absence of hypoxemia. For example, anemic hypoxia caused by reduced or dysfunctional hemoglobins (e.g., anemia, blood loss, carbon monoxide poisoning), histotoxic hypoxia caused by tissue dysfunction (e.g., cyanide poisoning), and circulatory hypoxia caused by perfusion defects (e.g., ↓cardiac output) may show normal PaO_2 measurements (Shapiro et al., 1991).

Hypoxemia. Hypoxemia is reduced oxygen in the blood. The PaO_2 from arterial blood gases is commonly used to evaluate a patient's oxygenation status. Since PaO_2 is a measurement of dissolved oxygen in the plasma, it does not represent the portion of oxygen carried by the hemoglobin. For precise assessment, arterial oxygen content (CaO_2) measured by co-oximetry should be used because it includes the oxygen combined with hemoglobin and the oxygen dissolved in the plasma.

When PaO_2 is used for oxygenation assessment, Table 1-8 may be used for interpretation of an adult's oxygenation status.

✓ Hypoxia can occur with a normal PaO_2.

Hypoxia. Hypoxia is reduced oxygen in the body organs and tissues. While hypoxemia and hypoxia are two terms sometimes used interchangeably, it is important to understand that hypoxia can occur with a normal PaO_2. Two examples illustrate this point. Cyanide poisoning causes histotoxic hypoxia in which the tissues cannot carry out aerobic metabolism. Anemia causes a low oxygen content (CaO_2) while the PaO_2 is often normal. Since PaO_2 measures the oxygen tension of the plasma only, it cannot be used for the assessment of histotoxic or anemic hypoxia. CaO_2 should be measured and used to assess the oxygenation status of a patient. In addition to the PaO_2 and CaO_2 measurements, hypoxia produces clinical signs (e.g., cyanosis, tachycardia, diaphoresis) that may be used as a secondary assessment tool.

✓ $CaO_2 = (Hb \times 1.34 \times SaO_2) + (PaO_2 \times 0.003)$

✓ Oxygenation failure may develop when severe hypoxemia ($PaO_2 < 40$ mm Hg) does not respond to a moderate to high level (50% to 100%) of supplemental oxygen.

Signs of Oxygenation Failure and Hypoxia. In most clinical situations, hypoxemia is readily corrected by a moderate amount of supplemental oxygen. Oxygenation

TABLE 1-8 Interpretation of Oxygenation Status Using PaO_2 at Different P_B

Hypoxemia Status	PaO_2 (P_B = 760 mm Hg)	PaO_2 (P_B = 630 mm Hg)
Normal	80 to 100 mm Hg	60 to 79 mm Hg
Mild	60 to 79 mm Hg	50 to 59 mm Hg
Moderate	40 to 59 mm Hg	40 to 49 mm Hg
Severe	Less than 40 mm Hg	Less than 40 mm Hg

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failure may develop when severe hypoxemia ($PaO_2 < 40$ mm Hg) does not respond to moderate to high levels (50% to 100%) of supplemental oxygen.

✓ The important clinical signs of oxygenation failure and hypoxia include hypoxemia, dyspnea, tachypnea, tachycardia, and cyanosis.

The important clinical signs of oxygenation failure and hypoxia include hypoxemia, dyspnea, tachypnea, tachycardia, and cyanosis (Rochester, 1993). In addition, patients often appear to have shortness of breath and may become disoriented. These signs are usually readily available in the medical records or at the bedside. They should be used in conjunction with laboratory results during "routine" ventilator rounds to assess the patient so that appropriate action may be taken.

CLINICAL CONDITIONS LEADING TO MECHANCIAL VENTILATION

✓ Mechanical ventilation is often used to support ventilatory or oxygenation failure.

Mechanical ventilation is often used to support ventilatory or oxygenation failure. Failure to ventilate or oxygenate adequately may be caused by pulmonary or nonpulmonary conditions. For example, adult respiratory distress syndrome is a pulmonary condition commonly associated with mechanical ventilation and mortality. Many nonpulmonary conditions (e.g., neuromuscular disease, acute brain injury) also contribute to the need for mechanical ventilation (Pierson, 2002; Kelly et al., 1993).

These pulmonary and nonpulmonary conditions often lead to a combination of deadspace ventilation, V/Q mismatch, shunt, diffusion defect, ventilatory failure, and oxygenation failure. For logical discussion and ease of patient management, they are separated into three distinct groups: (1) depressed respiratory drive (e.g., drug overdose), (2) excessive ventilatory workload (e.g., airflow obstruction), and (3) failure of ventilatory pump (e.g., chest trauma).

Depressed Respiratory Drive

✓ Depressed or insufficient respiratory drive may lead to ventilatory and oxygenation failure.

Depressed or insufficient respiratory drive may lead to a decrease in tidal volume, frequency, or both. These patients may have normal pulmonary function but the respiratory muscles do not have adequate neuromuscular impulses to function properly. Mechanical ventilation is used to support these patients until the cause of insufficient respiratory drive has been reversed.

Table 1-9 lists the clinical conditions that may lead to a depressed respiratory drive. They are drug overdose (Parsons, 1994), acute spinal cord injury (Bach, 1991), acute brain injury (Pierson, 2002), neurologic dysfunction (Kelly et al., 1993), sleep disorders, and compensation for metabolic alkalosis (Greene et al., 1994).

Excessive Ventilatory Workload

✓ Excessive ventilatory workload may lead to muscle fatigue and ventilatory/oxygenation failure.

Ventilatory workload is influenced by many clinical conditions (Table 1-10). When it exceeds the patient's ability to carry out the workload, ventilatory and

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TABLE 1-9 Causes of Depressed Respiratory Drive

Type of Respiratory Drive Depression	Clinical Conditions
Drug overdose	Central hypoventilation (narcotics, alcohol, sedatives) Acute respiratory insufficiency (cocaine, heroin, methadone, propoxyphene, phenothiazines, alcohol, barbiturates) Severe pulmonary complications (poisons and toxins such as paraquat, petroleum distillates, organophosphates, mushrooms of Amanita genus, hemlock, botulism)
Acute spinal cord injury	Respiratory paralysis (tetraplegic with injury at C1-C3 level)
Head trauma	Abnormal respiratory patterns (apnea, tachypnea, Cheyne-Stokes respiration, apneustic breathing, ataxic breathing) Neurogenic pulmonary edema (increase in intracranial pressure) Delayed pulmonary dysfunction (intrapulmonary shunt, increased pulmonary vascular resistance, V/Q mismatch)
Neurologic dysfunction	Coma Cerebral vascular accident (stroke) Altered mental status (hypoxic brain)
Sleep disorders	Sleep apnea (central, obstructive, mixed) Sleep deprivation
Metabolic alkalosis	Hypoventilation to compensate for elevated pH in metabolic alkalosis

oxygenation failure ensues and mechanical ventilation becomes necessary. The ventilatory workload is increased in the presence of severe airflow obstruction (Blanch et al., 2005), increased deadspace ventilation (Greene et al., 1994), acute lung injury (Kraus et al., 1993), congenital heart disease (DiCarlo et al., 1994), cardiovascular decompensation, shock (Hinson et al., 1992), increased metabolic rate, and decreased lung and chest wall compliance (Greene et al., 1994).

✓ Failure of the ventilatory pump may lead to an increased work of breathing and to eventual ventilatory and oxygenation failure.

Failure of Ventilatory Pump

Failure of the ventilatory pump is the structural dysfunction of the respiratory system to include the lung parenchyma, respiratory muscles, and thoracic skeletal

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TABLE 1-10 Causes of Excessive Ventilatory Workload

Type	Clinical Conditions
Acute airflow obstruction	Status asthmaticus Epiglotittis COPD
Deadspace ventilation	Pulmonary embolism Decrease in cardiac output Emphysema
Congenital heart disease	Hypoplastic left heart syndrome Tetralogy of Fallot Persistent pulmonary hypertension
Cardiovascular decompensation	Decreased cardiac output V/Q mismatch Deadspace ventilation
Shock	Blood loss Peripheral vasodilation Congestive heart failure
Increased metabolic rate	Fever Increased work of breathing
Drugs	Acute pulmonary edema (narcotics, salicylates, nonsteroidal anti-inflammatory agents, naloxone, thiazide diuretics, contrast media, insulin) Bronchospasm (salicylates, nonsteroidal anti-inflammatory agents, hydrocortisone, beta-blockers, neuromuscular blocking agents, contrast media)
Decreased compliance	Acute lung injury, ARDS, IRDS Atelectasis Tension pneumothorax Postthoracic surgery Obesity Diaphragmatic hernia

structures. If uncorrected, this condition may lead to increased work of breathing and eventual ventilatory and oxygenation failure.

Table 1-11 lists some clinical examples of conditions that may lead to ventilatory pump failure. They include chest trauma, prematurity (Watchko et al., 1994), electrolyte imbalance (Freeman et al., 1993), and problems in geriatric patients (Krieger, 1994).

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TABLE 1-11 Causes of Ventilatory Pump Failure

Type	Clinical Conditions
Chest trauma	Flail chest Tension pneumothorax
Premature birth	Idiopathic respiratory distress syndrome
Electrolyte imbalance	Hyperkalemia
Geriatric patients	Fatigue of respiratory muscles

SUMMARY

Mechanical ventilation is used for many different clinical conditions. Essentially all uses of mechanical ventilation are targeted toward patients who fail to ventilate or oxygenate adequately. Ventilatory and oxygenation failure may be due to the adverse changes in a patient's physiologic functions (i.e., depressed respiratory drive, excessive ventilatory workload, and failure of ventilatory pump). These abnormal physiologic functions should be identified early on so that the indications for mechanical ventilation are clearly delineated.

While mechanical ventilation does not treat any ventilation or oxygenation abnormalities per se, it is a useful adjunct to support the gas exchange function until effective spontaneous breathing or oxygenation is restored.

Self-Assessment Questions

1. Airway resistance may be increased in all of the following clinical conditions *except*:

   A. airway obstruction.
   B. endotracheal tube with small internal diameter.
   C. condensation in ventilator circuit.
   D. tachycardia.

2. A mechanically ventilated patient has an increased airflow resistance due to unknown reasons. In assessing the patient-ventilator system, the therapist should see ___ on the pressure-volume (P-V) display.

   A. widening (bowing) of the P-V loop
   B. narrowing of the P-V loop
   C. shifting of the P-V slope toward the volume axis
   D. shifting of the P-V slope toward the pressure axis

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3. Static compliance is primarily affected by a patient's ___ whereas the dynamic compliance is primarily affected by a patient's ___.

   A. elastic property of the lungs, airflow resistance
   B. elastic property of the lungs, minute ventilation
   C. airflow resistance, elastic property of the lungs
   D. airflow resistance, minute ventilation

4. The slope of a pressure-volume (P-V) loop is shifted toward the pressure axis. This condition is consistent with a(n):

   A. increase in compliance.
   B. increase in conductance.
   C. decrease in compliance.
   D. decrease in airflow resistance.

5. The most recent blood gas report shows that a patient is hypoventilating ($PaCO_2 = 65$ mm Hg). The physician asks the therapist to improve the patient's alveolar ventilation by making changes to the ventilator settings. The therapist should:

   A. decrease the tidal volume.
   B. increase the mechanical deadspace on the ventilator circuit.
   C. increase the frequency.
   D. increase the F_IO_2.

6. Hypoventilation is characterized by a(n):

   A. increased PaO_2.
   B. increased pH.
   C. increased $PaCO_2$.
   D. B and C only.

7. Ventilation/perfusion (V/Q) mismatch is common in lung diseases. For example, a low V/Q ratio may be seen in ___ and a high V/Q ratio in ___.

   A. atelectasis, pulmonary embolism
   B. pulmonary embolism, atelectasis
   C. atelectasis, airway obstruction
   D. airway obstruction, atelectasis

8. Which of the following causes of hypoxemia is *least* likely to be treated successfully by oxygen therapy alone?

   A. hypoventilation
   B. V/Q mismatch
   C. intrapulmonary shunting
   D. low P_IO_2

9. In managing a critically ill patient who has been on the ventilator for six days, the physician asks the therapist to calculate the estimated shunt percent using only one arterial blood gas sample. The therapist should use the equation below:

   A. Estimated $Q_{SP}/Q_T = (CcO_2 - CaO_2) / [5 + (CcO_2 - CaO_2)]$
   B. Estimated $Q_{SP}/Q_T = (CcO_2 - CaO_2) / [3.5 + (CcO_2 - CaO_2)]$
   C. Classic $Q_{SP}/Q_T = (CcO_2 - CaO_2) / (CcO_2 + CvO_2)$
   D. B or C only

10 to 13. Match the types of gas diffusion problem with the clinical conditions affecting the diffusion rate.

Types of Diffusion Problem	Clinical Conditions
10. Decreased O_2 pressure gradient	A. Emphysema
11. Thickening of A-C membrane	B. High altitude
12. Decreased surface area of A-C membrane	C. Tachycardia
13. Insufficient time for diffusion	D. Pulmonary edema

14. The ABG report for an abdominal postoperative patient shows respiratory acidosis with severe hypoxemia. In order to determine whether *hypoxia* is present, the therapist should evaluate all of the following *except*:

    A. $PaCO_2$.
    B. heart rate.
    C. color of skin.
    D. spontaneous frequency.

15 to 20. Ventilatory and oxygenation failure may occur when the respiratory drive is diminished. Match the types of respiratory depression with the respective clinical conditions.

Types of Depression	Clinical Conditions
15. Drug overdose	A. Altered mental status (hypoxic brain)
16. Acute spinal cord injury	B. Neurogenic pulmonary edema (increase of intracranial pressure)
17. Head trauma	C. Sleep apnea (central, obstructive, mixed)
18. Neurologic dysfunction	D. Hypoventilation to compensate for elevated pH
19. Sleep disorders	E. Respiratory paralysis (quadriplegic with injury at C1-C3 level)
20. Metabolic alkalosis	F. Narcotic and sedative use

21. Excessive and prolonged increase in the patient's respiratory workload may lead to fatigue of the ___ muscles. If uncorrected, ___ failure is the likely end result.

    A. heart, ventilatory and oxygenation
    B. heart, congestive heart
    C. respiratory, ventilatory and oxygenation
    D. respiratory, congestive heart

Answers to Self-Assessment Questions

1. D
2. A.
3. A.
4. C.
5. C.
6. C.
7. A.
8. C.
9. B.
10. B.
11. D.
12. A.
13. C.
14. A.
15. F.
16. E.
17. B.
18. A.
19. C.
20. D.
21. C.

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Chapter 2

Effects of Positive Pressure Ventilation

David W. Chang
Terry S. LeGrand

Outline

- Introduction
- Pulmonary Considerations
  - *Spontaneous Breathing*
  - *Positive Pressure Ventilation*
  - *Airway Pressures*
  - *Compliance*
- Cardiovascular Considerations
  - *Mean Airway Pressure and Cardiac Output*
  - *Decrease in Cardiac Output and O_2 Delivery*
  - *Blood Pressure Changes*
  - *Pulmonary Blood Flow and Thoracic Pump Mechanism*
- Hemodynamic Considerations
  - *Positive Pressure Ventilation*
  - *Positive End-Expiratory Pressure*
- Renal Considerations
  - *Renal Perfusion*
  - *Indicators of Renal Failure*
  - *Effects of Renal Failure on Drug Clearance*
- Hepatic Considerations
  - *PEEP and Hepatic Perfusion*
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- Nutritional Considerations
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  - *Nutritional Support*
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  - *Hyperventilation*
  - *Ventilatory and Oxygenation Failure*
  - *Indicators of Neurologic Impairment*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References

Key Terms

- central venous pressure (CVP)
- continuous positive airway pressure (CPAP)
- gastrointestinal (GI)
- hepatic perfusion
- intra-abdominal pressure (IAP)
- mean airway pressure (mPaw)
- oxygen delivery
- peak inspiratory pressure (PIP)
- positive end-expiratory pressure (PEEP)
- positive pressure ventilation
- pressure-controlled ventilation
- pulmonary artery pressure (PAP)
- pulmonary capillary wedge pressure (PCWP)
- renal perfusion
- stroke volume
- thoracic pump mechanism
- total parenteral nutrition (TPN)
- volume-controlled ventilation

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Describe the mechanism of ventilation using negative and positive airway pressures.
- Describe how positive pressure ventilation affects the airway pressure and compliance.
- List the effects of positive pressure ventilation on the following systems or parameters: cardiovascular, hemodynamic, renal, hepatic, abdominal, gastrointestinal, nutritional, and neurologic.
- Describe the effects of renal and hepatic impairments on drug clearance and therapeutic dose.

INTRODUCTION

Positive pressure ventilation is an essential life support measure in the intensive care and extended care environments. The physiologic effects of positive pressure ventilation have complex interactions with the lungs and other organ systems. Some of these physiologic effects are beneficial, while others may cause complications. This chapter discusses the effects of positive pressure ventilation and its side effects on the major organ systems of the body.

PULMONARY CONSIDERATIONS

This section compares the physiologic differences between spontaneous breathing and positive pressure ventilation. Two of the major effects are the changes in airway pressure and compliance.

Spontaneous Breathing

During negative pressure ventilation, pressures in the airways, alveoli, and pleura are *decreased* during inspiration.

During spontaneous ventilation, the diaphragm and other respiratory muscles create gas flow by lowering the pleural, alveolar, and airway pressures. When alveolar and airway pressures drop below atmospheric pressure, air flows into the lungs. Negative pressure ventilation uses this principle by creating a negative pressure on the chest wall. When negative pressure is used for ventilation, the pressures in the airways, alveoli, and pleura are decreased during inspiration. Table 2-1 shows the relationship between barometric pressure (P_B) and alveolar pressure (P_{ALV}) during spontaneous breathing.

The pressure readings in Table 2-1 are for illustration purposes only. The barometric pressure is assigned 0 cm H_2O for easy comparison of pressure changes during spontaneous breathing. (Wilkins et al., 2008).

Positive Pressure Ventilation

During **positive pressure ventilation**, gas flow is delivered to the lungs under a positive pressure gradient (i.e., airway pressure is greater than alveolar pressure). Under normal conditions, the tidal volume delivered to the lungs is directly related to the positive pressure when **pressure-controlled ventilation** is used. In **volume-controlled ventilation**, the level of positive pressure is dependent on the mechanical tidal volume, as well as the patient's compliance and airflow resistance.

When positive pressure is used for ventilation, the pressures in the airways, alveoli, and pleura are increased during inspiration. Table 2-2 shows the relationship between inspiratory pressure (P_I) and alveolar pressure (P_{ALV}) during positive

positive pressure ventilation: Mechancial ventilation in which the volume is delivered by a positive pressure gradient (i.e., airway pressure higher than alveolar pressure).

pressure-controlled ventilation: Mode of ventilation in which a preset peak inspiratory pressure is used to provide ventilation. The delivered volume during this mode of ventilation is affected by the changing compliance and resistance.

volume-controlled ventilation: Mode of ventilation in which a preset tidal volume is used to provide ventilation. The airway pressures during this mode of ventilation are affected by the changing compliance and resistance.

TABLE 2-1 Relationship of Barometric Pressure (P_B) and Alveolar Pressure (P_{ALV}) during Spontaneous Breathing

Spontaneous Breathing	P_B (cm H_2O)	P_{ALV} (cm H_2O)	ΔP	Flow
Inspiration	0	−5	−5	Into lungs
End-inspiration	0	0	0	None
Expiration	0	+5	+5	Out of lungs
End-expiration	0	0	0	None

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TABLE 2-2 Relationship of Inspiratory Pressure (P_I) and Alveolar Pressure (P_{ALV}) during Positive Pressure Ventilation

Positive Pressure Ventilation	P_I (cm H_2O)	P_{ALV} (cm H_2O)	ΔP	FLOW
Inspiration	20	0	+20	Into lungs
End-inspiration	20	20	0	No flow
Expiration	0	20	−20	Out of lungs
End-expiration	0	0	0	No flow

pressure ventilation. The pressure readings in Table 2-2 are for illustration purposes only. The barometric pressure is assigned 0 cm H_2O for easy comparison of pressure changes during positive pressure ventilation.

Under normal conditions, the pressure gradient and tidal volume are directly related. During pressure-controlled ventilation, a higher peak inspiratory pressure typically results in a larger tidal volume. However, there are some exceptions to this relationship. (Table 2-3).

Airway Pressures

peak inspiratory pressure (PIP): Maximum pressure measured during one respiratory cycle, usually at end-inspiration.

During pressure-controlled ventilation, the **peak inspiratory pressure (PIP)** is preset according to the estimated tidal volume requirement of a patient. The inspiratory phase terminates once the preset pressure is reached. For this reason, the patient may receive a smaller volume when the preset pressure is reached prematurely, as would happen under conditions of low compliance or high airway resistance.

During volume-controlled ventilation, the tidal volume is preset and the pressure used by the ventilator to deliver this preset volume is variable. The PIP at

TABLE 2-3 Conditions That Limit the Volume Delivered by Positive Pressure Ventilation

Conditions	Examples
Peak inspiratory pressure reached too soon	Airway obstruction Kinking of ET tube Bronchospasm Low lung compliance Pressure limit set too low
Unable to reach peak inspiratory pressure	ET tube cuff leak Ventilator circuit leak

end-inspiration is higher under conditions of low compliance or high airway resistance. On the other hand, the PIP is lower under conditions of high compliance or low airway resistance.

In positive pressure ventilation, airway pressures (including PIP and **mean airway pressure [mPaw]**) are directly related to the tidal volume, airway resistance, and peak inspiratory flow rate and inversely related to compliance.

These airway pressures (and lung volumes) have a direct impact on the intrathoracic pressure, blood flow, and blood pressure. Indirectly, they can affect the functions of major organ systems as they depend on adequate blood flow and perfusion.

mean airway pressure (mPaw): Average pressure within the airway during one complete respiratory cycle. It is directly related to the inspiratory time, respiratory frequency, peak inspiratory pressure, and positive end-expiratory pressure (PEEP).

Compliance

In lungs with normal compliance, about 50% of the airway pressure is transmitted to the thoracic cavity. In noncompliant or stiff lungs (e.g., atelectasis, ARDS), the pressure transmitted to the thoracic cavity is much less due to the dampening effect of the nonelastic lung tissues. For this reason, high levels of PIP or positive end-expiratory pressure (PEEP) may be required to ventilate and oxygenate patients with low compliance. The decrease in cardiac output due to excessive PIP or PEEP is less severe than that if the same pressures are applied to lungs with normal or high compliance (Perkins et al., 1989).

CARDIOVASCULAR CONSIDERATIONS

Mechanical ventilation creates airflow by generating a pressure gradient. In turn, the pressures in the airways, thoracic cage, and pulmonary blood vessels are altered. In a clinical setting, the cardiovascular functions should be evaluated and monitored to prevent the adverse effects of positive pressure ventilation on the heart and blood vessels.

Mean Airway Pressure and Cardiac Output

Positive pressure ventilation increases mPaw and decreases cardiac output. Regardless of the mode of ventilation, a higher mPaw usually results in a lower cardiac output (Perkins et al., 1989). Since mPaw is a function of inspiratory time, respiratory frequency, peak inspiratory pressure, and **positive end-expiratory pressure (PEEP)**, these four parameters should be kept to a minimum in order to keep the mPaw at the lowest level possible.

In comparing **continuous positive airway pressure (CPAP)** and PEEP, PEEP exerts a more negative effect on the cardiac output as it raises the mPaw (and PIP) proportionally. The effect of PEEP can be detrimental to the cardiac output because PEEP is the end-expiratory pressure used in addition to positive pressure ventilation—whereas, in CPAP, the pressure includes only the positive airway pressure during spontaneous breathing (Figure 2-1).

positive end-expiratory pressure (PEEP): PEEP is an airway pressure strategy in ventilation that increases the end-expiratory or baseline airway pressure to a value greater than atmospheric pressure. It is used to treat refractory hypoxemia caused by intrapulmonary shunting.

continuous positive airway pressure (CPAP): The end-expiratory pressure applied to the airway of a spontaneously breathing patient.

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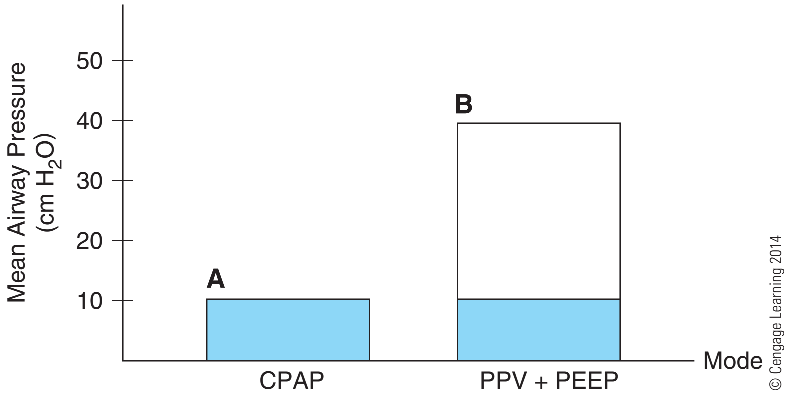

FIGURE 2-1 Comparison of mean airway pressure between (A) CPAP and (B) PEEP. The mean airway pressure is higher in (B) because PEEP (10 cm H_2O) is used in addition to positive pressure ventilation.

✔ A decreased venous return (or filling of ventricles) leads to a reduction in stroke volume and cardiac output.

Decrease in Cardiac Output and O_2 Delivery

Use of positive pressure ventilation can reduce the amount of oxygen available to the body. An increase in positive airway pressure generally causes a higher intrathoracic pressure. In turn, this pressure is transmitted to the airways and alveoli, as well as to the mediastinum, and great vessels. Compression of great vessels can lead to decreased venous return to the heart (Marini, 2004). A decreased venous return (or filling of ventricles) leads to a reduction in **stroke volume** and cardiac output.

stroke volume: Blood volume output delivered by one ventricular contraction.

oxygen delivery: Total amount of oxygen carried by blood. It is the product of O_2 content and cardiac output.

Since O_2 delivery is the product of O_2 content and cardiac output, reduction in stroke volume and cardiac output results in a decrease in **oxygen delivery**. As shown in the equation below and Figure 2-2, decreased cardiac output reduces O_2 delivery.

$$O_2 \text{ Content} \times \downarrow\text{Cardiac Output} = \downarrow O_2 \text{ Delivery}$$

Blood Pressure Changes

✔ During spontaneous inspiration, a transient decrease of arterial blood pressure is called *pulsus paradoxus*.

✔ A significant reverse pulsus paradoxus (increase of systolic pressure >15 mm Hg) during positive pressure ventilation is a sensitive indicator of hypovolemia.

During spontaneous inspiration, there is a transient decrease of arterial blood pressure. In cardiac tamponade or acute asthma exacerbation, this transient decrease in systolic blood pressure becomes exaggerated (>10 mm Hg decrease), the condition is called *pulsus paradoxus* (Abu-Hilal et al., 2010).

During positive pressure ventilation, reverse pulsus paradoxus is observed in which the arterial blood pressure is slightly higher than that measured during spontaneous breathing. During positive pressure ventilation, pressures measured in the aorta, left atrium, pulmonary artery, and right atrium are also slightly higher than those measured during spontaneous ventilation. Positive pressure ventilation also displaces the ventricle walls inward during systole. This movement enhances ventricular emptying leading to a slight rise in systolic pressure. The mechanism of reverse pulsus paradoxus appears to be a reduction in left ventricular afterload (Abel et al., 1987). A significant reverse pulsus paradoxus (increase of systolic pressure >15 mm Hg) during positive pressure ventilation is a sensitive indicator of hypovolemia. For patients with cardiopulmonary disease or compromised cardiovascular reserve, positive pressure ventilation and PEEP may further lower the venous return and compromise the cardiovascular functions (Abel et al., 1987; Jithesh, 2006; Shekerdemian et al., 1999).

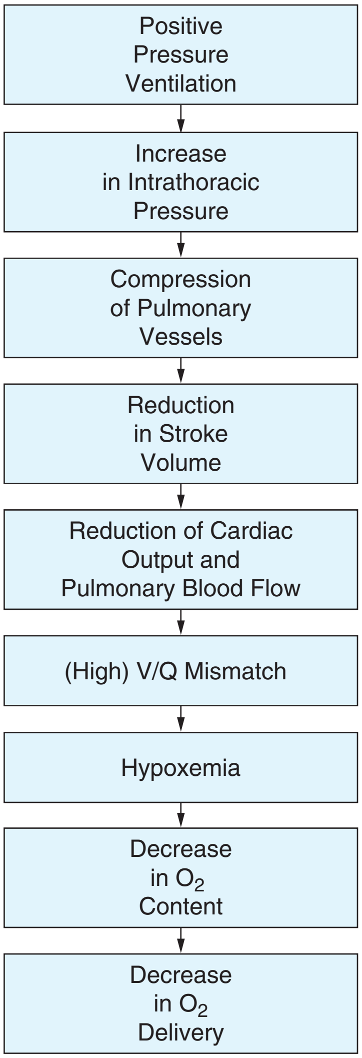
Positive Pressure Ventilation
Increase in Intrathoracic Pressure
Compression of Pulmonary Vessels
Reduction in Stroke Volume
Reduction of Cardiac Output and Pulmonary Blood Flow
(High) V/Q Mismatch
Hypoxemia
Decrease in O_2 Content
Decrease in O_2 Delivery

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FIGURE 2-2 Positive pressure ventilation leads to a decrease in O_2 delivery.

Pulmonary Blood Flow and Thoracic Pump Mechanism

During positive pressure ventilation, intrathoracic pressure changes according to the pressure transmitted across the lung parenchyma. In turn, changes in intrathoracic pressure can affect the pulmonary blood flow entering and leaving the ventricles.

Left ventricle. In the left ventricle, the effect of an increase in lung volume on pulmonary venous blood flow is dependent on the relative state of filling of the pulmonary circulation. In patients who are *hypotensive*, an increase in tidal volume causes a decrease in pulmonary venous return to the left ventricle (Figure 2-3) (Pinsky, 1990).

In *hypertensive* patients, use of large tidal volumes increases venous return to the left ventricle (Figure 2-4) (Pinsky, 1990). This is because compression of pulmonary blood vessels is minimal in hypertensive conditions. It is also due in part to the **thoracic pump mechanism** where the blood flow from right to left ventricle is enhanced during the expiratory phase of positive pressure ventilation (DiCarlo et al., 1994).

thoracic pump mechanism: Alternations in pulmonary blood flow caused by changes in intrathoracic pressure during positive pressure ventilation. In hypotensive conditions, positive pressure ventilation decreases the blood flow to the left heart. In hypertensive conditions, this mechanism enhances the outflow of blood from the right ventricle and into the left heart.

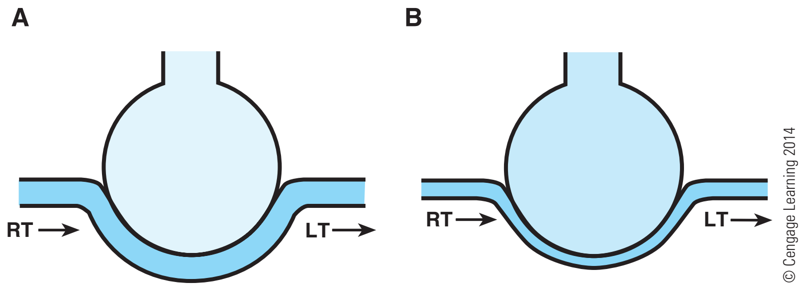

FIGURE 2-3 In hypotensive conditions, positive pressure ventilation decreases the blood flow to left heart. (A) Spontaneous breathing; (B) Positive pressure ventilation causes compression of the pulmonary blood vessels in hypotensive conditions.

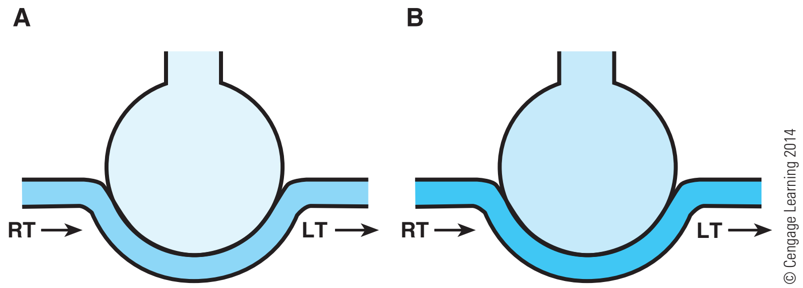

FIGURE 2-4 In hypertensive conditions, positive pressure ventilation increases the blood flow to left heart in part due to the thoracic pump mechanism (see text). (A) Spontaneous breathing; (B) Positive pressure ventilation does not cause significant compression of the pulmonary blood vessels in hypertensive conditions.

Right ventricle. In the right ventricle, high airway pressures and large tidal volumes used in positive pressure ventilation stretch and compress the pulmonary blood vessels and limit their capacity to hold blood volume. During expiration, the pulmonary vessels, no longer under high pressure and large tidal volumes, are free to fill to their holding capacity with the blood leaving the right ventricle. This thoracic pump mechanism facilitates the outflow of blood from the right ventricle (Figure 2-5) (DiCarlo et al., 1994).

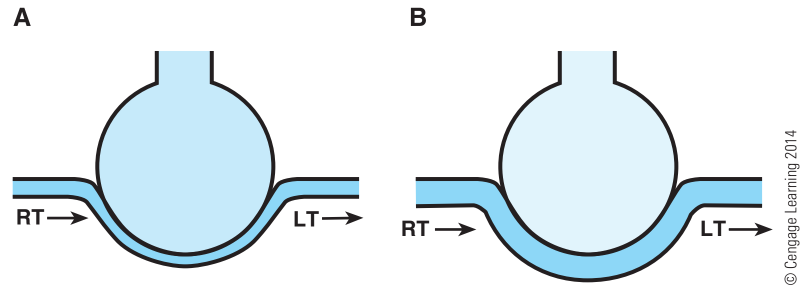

FIGURE 2-5 Thoracic pump mechanism. (A) During the inspiratory phase of positive pressure ventilation, the pulmonary blood vessels are compressed and the blood flow from right to left ventricle is decreased. (B) During the expiratory phase, the pulmonary blood vessels are no longer under alveolar compression and the blood flow from right to left ventricle is increased.

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In children with right ventricular dysfunction, high positive pressure (up to 40 cm H_2O) and large tidal volumes (20 to 30 mL/kg) may reduce the workload of the right heart by the action of the thoracic pump mechanism (DiCarlo et al., 1994).

HEMODYNAMIC CONSIDERATIONS

One of the major adverse effects of mechanical ventilation is the changes in a patient's hemodynamic status. The major hemodynamic measurements affected by positive pressure ventilation include **central venous pressure (CVP)** and **pulmonary artery pressure (PAP)**. The **pulmonary capillary wedge pressure (PCWP)** is not affected to a great extent because of the capability of the systemic venous circulation to compensate or regulate changing blood pressure and volume.

central venous pressure (CVP): Pressure measured in the vena cava or right atrium. It reflects the status of blood volume in the systemic circulation. Right ventricular preload.

pulmonary artery pressure (PAP): Pressure measured in the pulmonary artery. It reflects the volume status of the pulmonary artery and the functions of the ventricles. Right ventricular afterload.

pulmonary capillary wedge pressure (PCWP): Pressure measured in the pulmonary artery with a balloon inflated to stop pulmonary blood flow. It reflects the volume status and functions of the left heart. Left ventricular preload.

Positive Pressure Ventilation

Positive pressure ventilation causes an increase in intrathoracic pressure and compression of the pulmonary blood vessels. Partial recovery is observed during the expiratory phase. It is estimated that 15% to 20% of pulmonary blood volume is shifted to the systemic circulation at a tidal volume of 1 L. An increase in intrathoracic pressure and compression of the pulmonary blood vessels causes an overall decrease in ventricular output, stroke volume, and pressure readings (Versprille, 1990).

Table 2-4 shows the general effects of positive pressure ventilation on hemodynamic measurements. It is essential to remember that the severity of these hemodynamic changes is dependent on the level of airway pressures, lung volume, and compliance characteristics of the patient.

✓ Positive pressure ventilation causes an increase in intrathoracic pressure and compression of the pulmonary blood vessels leading to an overall decrease in ventricular output, stroke volume, and pressure readings.

Positive End-Expiratory Pressure

Positive end-expiratory pressure (PEEP) is a modality used in conjunction with positive pressure ventilation. PEEP has a profound effect on the PAP and mPaw. In one study, when PEEP was initiated and increased to 15 cm H_2O over 90 sec, the CVP and PAP showed a drastic increase while the aortic pressure and cardiac output showed a significant decrease (Versprille, 1990). PEEP must be used with extreme care in a clinical setting because PEEP, in addition to positive pressure ventilation, can potentiate the reduction in cardiac output.

✓ PEEP increases CVP and PAP but decreases aortic pressure and cardiac output.

Table 2-5 outlines the general effects of PEEP on hemodynamic measurements. It is important to remember that PEEP is used in conjunction with positive pressure ventilation. For this reason, the hemodynamic changes may be different from those caused by positive pressure ventilation alone. The severity of these hemodynamic changes is also dependent on the lung volume and compliance.

The decrease in cardiac output due to positive pressure ventilation and PEEP can be managed by using appropriate intravascular volume expansion and positive inotropic support. A patient with adequate intravascular volume or one who receives a positive inotrope may have a smaller decline in cardiac output during positive pressure ventilation and PEEP (Perkins et al., 1989).

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TABLE 2-4 Effects of Positive Pressure Ventilation on Hemodynamic Measurements

Pressure or Volume Changes	Notes
Increase in intrathoracic pressure	Positive pressure applied to the lungs causes compression of the lung parenchyma against the chest wall.
Decrease in pulmonary blood volume and *increase* in systemic blood volume	During the inspiratory phase of positive pressure ventilation, a fraction of the pulmonary blood volume is shifted to the systemic circulation. This does not increase the central venous pressure (CVP) because the systemic venous circulation can readily absorb this extra volume.
Decrease in venous return (CVP)	Higher intrathoracic pressure impedes systemic blood return to right ventricle.
Decrease in right ventricular stroke volume*	Decreased venous return to right ventricle leads to lower right ventricular output.
Decrease in pulmonary arterial pressure (PAP)	Decreased right ventricular stroke volume leads to lower blood volume (pressure) in the pulmonary arteries.
Decrease in filling pressures	Lower blood volume entering and leaving the ventricles.
Decrease in left ventricular stroke volume*	Decreased right ventricular stroke volume and pulmonary artery pressure lead to lower left ventricular input and output.

**NOTE: In the absence of compensation by increasing the heart rate, decrease in right and left ventricular stroke volumes generally leads to a decreased cardiac output.*

RENAL CONSIDERATIONS

Kidneys play an important role in eliminating wastes, clearance of certain drugs, and regulating fluid, electrolyte, and acid-base balance. The kidneys are highly vascular and at any one time receive about 25% of the circulating blood volume (Brundage, 1992). Because of these characteristics, they are highly vulnerable to decreases in blood flow, as it would occur during positive pressure ventilation.

Renal Perfusion

renal perfusion: Blood flow to the kidneys. It is decreased when blood volume or cardiac output is low.

When **renal perfusion** or perfusion of the glomeruli of the kidneys is decreased, filtration becomes less efficient (Baer et al., 1992). Subsequently, the urine output is decreased, as the kidneys try to correct the hypovolemic condition by retaining fluid. If hypoperfusion of the kidneys persists or worsens, renal failure may result.

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TABLE 2-5 Effects of Positive End-Expiratory Pressure on Hemodynamic Measurements

Pressure or Cardiac Output Change	Notes
Increase in pulmonary artery pressure (PAP)	PEEP and positive pressure applied to the lungs cause significant compression of pulmonary blood vessels.
Increase in central venous pressure (CVP)	Increase in PAP causes a higher right ventricular pressure and hinders blood return from systemic circulation to right heart. This causes backup of blood flow and increase in pressure in the systemic venous circulation.
Decrease in aortic pressure	This is due to the significant increase in intrathoracic pressure and significant *decrease* in left and right ventricular stroke volumes.
Decrease in cardiac output	This is due to the significant increase in intrathoracic pressure and significant *decrease* in left and right ventricular stroke volumes.

Indicators of Renal Failure

✓ Oliguria is defined as urine output < 400 mL in 24 hours (or <160 mL in 8 hours).

For adequate removal of body wastes, urine output must be above 400 mL in a 24-hour period. Decreased urine output is an early sign of renal insufficiency or failure. This condition is called oliguria and is defined as urine output less than 400 mL in 24 hours (or less than 160 mL in 8 hours) (Kraus et al., 1993). Other early signs of renal failure include elevation of serum blood urea nitrogen (BUN) and creatinine, products of nitrogen metabolism (King, 1994). The kidney is responsible for eliminating these nitrogenous wastes to prevent toxic accumulation in the body; thus an increase in serum levels of BUN and creatinine indicates compromised renal function. See Table 2-6 for other major serum indicators of renal failure.

Effects of Renal Failure on Drug Clearance

✓ Normal kidney functions include filtration of wastes, regulation of fluid, electrolyte, and acid-base balance, and clearance of some drugs.

Whenever the kidneys are not functioning properly, their performance is hindered. Renal dysfunctions can affect normal kidney functions to include filtration of wastes, regulation of fluid, electrolyte and acid-base balance, and clearance of some drugs.

Hypoperfusion. Hypoperfusion of the kidneys may affect the rate of drug clearance. This condition leads to a higher systemic concentration of drugs that rely on renal clearance. For example, the duration of neuromuscular blockade after short-term use of pancuronium or vecuronium may be prolonged in renal failure. The duration of muscle paralysis may be as long as 7 days after receiving vecuronium for more than

TABLE 2-6 Serum Indicators of Renal Failure

Serum Measurements	Normal	Renal Failure
Blood urea nitrogen (BUN)	10 to 20 mg/dL	Increased
Creatinine	0.7 to 1.5 mg/dL	Increased
BUN to creatinine ratio	10:1	Normal or increased
Creatinine clearance (male)	97 to 137 ml/min	Decreased
Glomerular filtration rate	90 to 120 mL/min/1.73 m^2	$<$15 mL/min/1.73 m^2
Potassium	3 to 5 mEq/L	Usually increased
Sodium	138 to 142 mEq/L	Usually decreased

✓ Hypoperfusion of the kidneys may affect the rate of drug clearance leading to a higher drug concentration in the circulation.

2 days (Hansen-Flaschen et al., 1993). The possibility of prolonged neuromuscular blockade can reduce a patient's lung mechanics and ability to be weaned from mechanical ventilation.

Glomerular Filtration Rate (GFR). Decreased renal function caused by positive pressure ventilation may also affect other drugs whose clearance is mainly dependent on the GFR of the kidneys. Glomerular filtration results from high pressure within the glomerulus or renal capillary. This is caused by differences in the tone of the afferent and efferent arterioles, the vessels that lead into and out of the glomerulus. The afferent arteriole is maintained in a somewhat dilated state relative to the efferent arteriole, which is always somewhat constricted. When blood flow to the kidney is normal, a hydrostatic pressure head causes the high rate of renal perfusion seen in the normovolemic state. When coupled with back pressure from the partially constricted efferent arteriole, pressures within the glomerulus are maintained at an elevated state and are responsible for its ultrafiltration function. When renal perfusion drops, the pressure causing glomerular filtration decreases, leading to a decrease in filtration.

Examples of drugs that are eliminated by this mechanism include digoxin, vancomycin, beta-lactam antibiotics, and the aminoglycosides (e.g., gentamycin, tobramycin) (Perkins et al., 1989). A decreased GFR or decreased creatinine clearance may lead to a higher concentration of these drugs in the circulation.

Tubular Secretion. Another group of drugs whose elimination could be reduced by a lower renal blood flow are drugs undergoing tubular secretion. Tubular secretion is the mechanism whereby substances are secreted from the blood via the peritubular capillaries into the renal tubule to become a part of the urine. Examples of drugs that are eliminated by this mechanism include digoxin, furosemide, procainamide, and some penicillins (Perkins et al., 1989). Decrease of renal tubular secretion causes a relatively higher concentration of these drugs in the circulation.

Reabsorption. The third group of drugs whose elimination could be decreased are those being reabsorbed at a higher rate. Reabsorption in the renal tubules is the mechanism whereby required substances that are filtered by the glomerulus are reclaimed by the cells lining the renal tubule and are ultimately reabsorbed into the blood. Some of these substances are reabsorbed down to an equal concentration gradient; thus an enhanced concentration gradient could lead to increased reabsorption.

As cardiac output is reduced by mechanical ventilation, renal blood flow and, thus, urine volume, are also reduced. As the urine becomes more concentrated, the drugs in the glomerular filtrate also become more concentrated. This causes an increase in the reabsorption gradient of the drugs in the filtrate. Some of the drugs used in critically ill patients include aminoglycosides, theophylline, and phenobarbital (Perkins et al., 1989). Decreased renal perfusion causes a higher reabsorption rate of these drugs back into the circulation.

HEPATIC CONSIDERATIONS

hepatic perfusion: Blood flow to the liver. It is decreased when the blood volume or cardiac output is low.

Hepatic perfusion accounts for about 15% of the total cardiac output. Positive pressure ventilation alone does not alter the blood flow to the liver to any significant degree. When PEEP is added to mechanical ventilation, the blood flow to the liver is noticeably reduced (Bonnet et al., 1982).

PEEP and Hepatic Perfusion

The rate of hepatic blood flow is inversely related to the level of PEEP. In one study, the hepatic blood flow decreased 3%, 12%, and 32% at PEEP of 10, 15, and 20 cm H_2O, respectively (Bonnet et al., 1982). The decrease in hepatic blood flow is solely caused by a reduction in cardiac output as a result of PEEP. This inference is made because the ratio of hepatic blood flow to cardiac output remains unchanged at 15% during mechanical ventilation without PEEP (Perkins et al., 1989).

✓ Impairment of liver function is likely when prothrombin time is >4 sec, bilirubin level is ≥50 mg/L, or albumin level is ≤20 g/L.

Indicators of Liver Dysfunction

Liver dysfunction may be monitored by measuring the prothrombin time and bilirubin and albumin levels (Kraus et al., 1993). Impairment of liver function is likely when coagulation time is increased (prothrombin time >4 sec over control, bilirubin level is increased (≥50 mg/L), or albumin level is decreased (≤20 g/L).

Effects of Decreased Hepatic Perfusion on Drug Clearance

✓ Hypoperfusion of the liver may affect the rate of drug clearance and lead to a higher drug concentration in the circulation.

A decrease in hepatic blood flow may diminish the drug clearance mechanism of the liver. Drugs most likely to be affected by changes in hepatic blood flow are agents whose clearance relies on the liver and its perfusion. Examples of such drugs commonly used in the intensive care unit include lidocaine, meperidine, propranolol, and verapamil (Perkins et al., 1989). When hepatic perfusion is inadequate, use of these drugs may lead to a relatively higher serum concentration due to diminished drug clearance.

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ABDOMINAL CONSIDERATIONS

intra-abdominal pressure (IAP): Pressure measured by a transducer via a transurethral bladder catheter.

Increases in **intra-abdominal pressure (IAP)** are related to clinical conditions such as bowel edema or obstruction and ascites. IAP may also be increased in procedures such as use of pneumatic antishock garments and surgical repair of abdominal wall hernias. When these patients are placed on mechanical ventilation, conditions that are conducive to an increase in IAP should be monitored to avert potential complications.

Effects of PEEP and Increased Intra-Abdominal Pressure

✓ Elevated intra-abdominal pressure transmits excessive pressure across the diaphragm to the heart and great vessels. In turn, this excess pressure leads to decreased cardiac output and decreased renal perfusion.

An elevated IAP transmits excessive pressure across the diaphragm to the heart and great vessels. In turn, this excessive pressure leads to decreased cardiac output (Cullen et al., 1989) and decreased renal perfusion (Harman et al., 1982). Excessive IAP also compresses the lungs and reduces the functional residual capacity (Burchard et al., 1985). It has been shown that use of PEEP on patients with elevated IAP may lead to cardiovascular, renal, and pulmonary dysfunction (Burchard et al., 1985; Cullen et al., 1989; Harman et al., 1982). These types of dysfunction are summarized in Table 2-7.

Use of high levels of PEEP (>15 cm H_2O) in the presence of high IAP (>20 mm Hg) requires caution because of potentiation of the pressures exerted on the heart and great vessels (Sussman et al., 1991). In patients with low pulmonary compliance (e.g., ARDS), the pressures transmitted to the heart and great vessels are not as severe as due to the dampening effects of the noncompliant lungs and chest wall.

TABLE 2-7 Effects of PEEP and Increased Intra-Abdominal Pressure

System	Effects
Cardiovascular	Increased peripheral vascular resistance Decreased compliance of ventricles Decreased cardiac output
Renal	Decreased renal perfusion Decreased glomerular filtration rate
Pulmonary	Decreased functional residual capacity Increased atelectasis Impaired gas exchange Increased V/Q mismatch and venous admixture

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GASTROINTESTINAL CONSIDERATIONS

gastrointestinal (GI): Organ system including the stomach and intestines.

✓ GI complications may be caused by a decrease of perfusion to the GI tract and medications that are commonly used in mechanically ventilated patients.

Positive pressure ventilation is associated with many organ-system complications, one of which involves the **gastrointestinal (GI)** tract. GI complications in patients who are being mechanically ventilated include erosive esophagitis, stress-related mucosal damage (SRMD), diarrhea, decreased bowel sounds, high gastric residuals, and constipation (Mutlu et al., 2000). These conditions are likely caused by a decrease of perfusion to the GI tract and medications commonly used during mechanical ventilation. Morphine sulfate is a medication commonly used for pain control. Some of the GI side effects of morphine include constipation and vomiting. Table 2-8 summarizes the factors that may adversely affect the normal GI functions (Mutlu et al., 2001).

NUTRITIONAL CONSIDERATIONS

Malnutrition in critically ill patients can create muscle fatigue, ventilatory insufficiency, and ventilatory failure. This sequence of events can lead to a need for mechanical ventilation. It can also make weaning from mechanical ventilation difficult or unsuccessful. Adequate nutritional support is therefore essential in the management of critically ill patients. However, excessive nutritional support is undesirable since it may cause excessive carbon dioxide production, as well as increased work of breathing in order to eliminate excessive CO_2 (van den Berg & Stam, 1988).

TABLE 2-8 Major Factors Adversely Affecting Normal GI Functions

Factors	Notes
Mechanical ventilation (especially with high levels of PEEP)	Increased intrathoracic pressure Increased right atrial pressure Reduced pressure gradient between mean systemic venous and right atrial pressures Reduced cardiac output and GI perfusion
Splanchnic hypoperfusion	Due to decreased mean arterial pressure and/or increased resistance in the GI vascular bed May lead to stress-related mucosal damage (SRMD)
Medications (e.g., opiates, sedatives, vasopressors, antibiotics)	Decreased GI motility Impaired venous return via venodilation and/or diminution of response to vasopressors

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Muscle Fatigue

✓ This caloric cost of breathing for COPD patients is about 10 times that of normal individuals (normal = 38 to 72 kcal/day) Over time, these abnormalities may cause fatigue of the respiratory muscles, and ventilatory failure with concurrent CO_2 retention and hypoxemia (Brown, 1994).

The work of breathing can be affected by mechanical aberrations such as changes in airway resistance and lung or chest wall compliance. In clinical conditions where there is a persistent increase in airway resistance (e.g., COPD) or reduction in compliance (e.g., atelectasis), the respiratory muscles must work strenuously to overcome the abnormal resistance and compliance. For instance, COPD patients use 430 to 720 kcal/day to carry out the work of breathing. This caloric cost of breathing for COPD patients is about 10 times that of normal individuals (normal = 38 to 72 kcal/day) (Brown, 1983) because of the increased work of breathing necessary to overcome the high airway resistance and V/Q abnormalities.

Other than the mechanical aberrations that can lead to increased work of breathing and eventual muscle fatigue, there are nonmechanical factors as well. Malnutrition is an example of a nonmechanical cause of muscle fatigue that may lead to ventilatory failure (Fiaccadori et al., 1991).

Table 2-9 shows the major mechanical and nonmechanical factors that may lead to reduced respiratory muscle efficiency and eventual muscle fatigue.

Diaphragmatic Dysfunction

✓ Prolonged positive pressure ventilation can induce diaphragmatic dysfunction.

Prolonged positive pressure ventilation can induce diaphragmatic dysfunction. For patients undergoing prolonged mechanical ventilation, atrophy of the diaphragm muscles can occur as a result of muscle proteolysis and a decrease in myofiber content. Furthermore, the loss of diaphragm force is time-dependent (Haitsma, 2011). For this reason, weaning from mechanical ventilation should be initiated as soon as feasible.

Nutritional Support

Adequate nutrition is a therapeutic necessity in order to provide and preserve inspiratory muscle strength and prevent ventilatory failure. Patients who have respiratory disorders are likely to lose weight due to increased work of breathing, decreased nutritional intake,

TABLE 2-9 Factors Leading to Respiratory Muscle Fatigue

Mechanical Factors	Nonmechanical Factors
High airway resistance	Malnutrition
Low lung compliance	Endocrine diseases (high metabolic rate)
Low chest wall compliance	Electrolyte disorders Drugs Persistent hypoxemia

(Data from Fiaccadori et al., 1991; Grassino et al., 1984; Rochester, 1986.)

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and infectious states causing increased metabolic rate. Most mechanically ventilated patients who are fed enterally do not receive their energy requirement mainly because of frequent interruptions in enteral feedings (O'Leary-Kelley et al., 2005).

Inadequate nutrition leads to protein catabolism and a loss of muscle performance (MacIntyre, 2001). Undernutrition in patients is found to deplete their stores of glycogen and protein in the diaphragm. In addition, COPD patients who have nutritional deficits may develop muscular dysfunction and peripheral muscle waste (Ambrosino et al., 2004). Since the diaphragm is the major respiratory muscle, loss of muscle mass in the diaphragm may reduce the efficiency of spontaneous ventilation.

Energy requirements for critically ill patients are normally computed by using the Harris-Benedict equation. This equation estimates the resting energy expenditure (REE) based on weight, height, age, and gender. The REE is not equivalent to the total number of calories required by a given patient. Rather, the patient's total calorie needs are estimated by multiplying the REE by an appropriate activity or stress factor. In ventilator patients who are hypermetabolic or hypercatabolic (i.e., infection, trauma, burns), a correction factor is included to allow for additional metabolic needs. It ranges from 1.2 to 1.6 times the REE for severe infection and 1.5 to 2.1 times the REE for burns. For most hospitalized patients under mild to moderate stress, the calorie requirement may be estimated by using 25 to 35 cal/Kg (Parrish et al., 2003).

Nutrition and the Work of Breathing

total parenteral nutrition (TPN): Complete nutritional support provided to the patient by any method (usually intravenous) other than the intestinal route.

Total parenteral nutrition (TPN) or hyperalimentation is a complete nutritional program provided to patients by any method (usually intravenous) other than the intestinal route. It is often used to support and supplement a patient's nutritional needs with a hypertonic solution consisting of amino acids, glucose, vitamins, electrolytes, and fat emulsion. When TPN is used, it is essential to keep the amount of dextrose (a carbohydrate) to a minimum, as it can cause lipogenesis and increase O_2 consumption and CO_2 production. Contribution to the total caloric needs by glucose should be in the range of 40% to 60% (Brown et al., 1984).

✓ Since fat emulsion provides 9.1 kcal/g, it provides maximum caloric intake with minimum CO_2 production.

Since hydrous dextrose generates 3.4 kcal/g and fat emulsion provides 9.1 kcal/g, fat is the ideal source of energy for patients who have restricted fluid intake. Fat also reduces CO_2 production, a by-product of glucose metabolism, thus reducing the work of breathing (Brown et al., 1983). A fat-based TPN should be considered for patients with significant or persistent CO_2 retention, as fat emulsion may provide maximum caloric intake with minimum CO_2 production.

It is important to note that the work of breathing is significantly increased in patients receiving high caloric intake by means of TPN. The increase in work of breathing is primarily due to increases in oxygen consumption and carbon dioxide production during TPN (van den Berg & Stam, 1988). Mechanical ventilation and weaning strategies must take this condition into account in order to provide adequate ventilatory support.

The increase in $\dot{V}CO_2$ causes a rise in $PaCO_2$, resulting in respiratory acidosis. Ventilatory failure can occur if the patient is unable to increase ventilation in

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proportion to the increase of CO_2 production. This is particularly important in patients with impaired ventilatory reserves.

NEUROLOGIC CONSIDERATIONS

Among many other monitoring systems that influence the respiratory drive, the central and peripheral chemoreceptors respond rapidly to the levels of CO_2, H^+, and O_2 in the blood. For this reason, the degree of ventilation (CO_2, H^+) and oxygenation (O_2) can affect the normal functions of the brain.

Hyperventilation

✓ Sustained hyperventilation of less than 24 hours causes respiratory alkalosis and reduces cerebral blood flow and intracranial pressure.

Carbon dioxide acts as a vasodilator in cerebral blood vessels. During mechanical ventilation, intentional hyperventilation is sometimes used to constrict these blood vessels, and thus minimize intracranial pressure in patients with head trauma. Sustained hyperventilation of less than 24 hours causes respiratory alkalosis, reducing cerebral blood flow and intracranial pressure. After 24 hours, the buffer systems of the body return the pH toward normal, negating the vasoconstrictor effect of controlled hyperventilation.

✓ 2, 3-bisphosphoglycerate (2, 3-BPG) is also known as 2,3-diphosphoglycerate (2, 3-DPG)

If hyperventilation is prolonged, cerebral tissue hypoxia may result due to the leftward shift of the oxyhemoglobin curve. A left shift causes higher oxygen affinity for hemoglobin but reduced oxygen release to tissues. Sustained hyperventilation also produces significant hypophosphatemia because of movement of phosphate into the cells. Hypophosphatemia interferes with cerebral tissue metabolism by reducing ATP stores and 2,3-BPG levels, which further increases the leftward shift of the oxyhemoglobin curve (Jozefowicz, 1989). Table 2-10 summarizes the neurologic changes in short-term ($<$24 hours) and sustained ($>$24 hours) hyperventilation.

TABLE 2-10 Neurologic Changes in Hyperventilation

Condition	Pathophysiologic Changes
Respiratory alkalosis ($<$24 hours)	Decreased cerebral blood flow Reduced intracranial pressure
Respiratory alkalosis (Prolonged $>$24 hours)	Leftward shift of oxyhemoglobin curve Increased O_2 affinity for hemoglobin Reduced O_2 release to tissues Cerebral tissue hypoxia Neurologic dysfunction Hypophosphatemia

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Ventilatory and Oxygenation Failure

Ventilatory and oxygenation failure has serious and detrimental effects on the central nervous system (CNS). Such failure may occur in patients on mechanical ventilation because of preexisting clinical conditions, making ventilation and oxygenation extremely difficult to accomplish in spite of high F_IO_2 and PEEP.

Abnormalities in ventilation and gas exchange can cause hypercapnia (increase in $PaCO_2$), respiratory acidosis (decrease in pH as a result of the increased $PaCO_2$), hypoxemia (decrease in PaO_2), secondary polycythemia (increase in red blood cell concentration and thus hemoglobin level), and electrolyte disturbances. These changes may lead to neurologic impairment.

Indicators of Neurologic Impairment

✓ Headache, mental status changes, motor disturbances, and ocular abnormalities may be signs of neurologic impairment.

When neurologic functions are impaired due to ventilatory and oxygenation failure, the patient may experience headache, mental status changes, motor disturbances, and ocular abnormalities (Jozefowicz, 1989).

The patient usually describes the headache as "pressure in the head," having a higher intensity during night and early morning hours. The headache is the result of cerebral vasodilation in response to hypoventilation and CO_2 retention during sleep.

Hypoxia, hypercapnia, and acidosis are responsible for the changes in a patient's mental status. Early mental disturbances include drowsiness, forgetfulness, and irritability. In severe or chronic cases of hypoxia and hypercapnia, stupor and coma may occur.

Hypercapnia may also cause muscle tremor and ocular abnormalities. Muscle tremor is the result of excessive stimulation of the sympathetic nervous system and catecholamine release from the adrenal medulla. Ocular abnormalities such as papilledema, swelling of the area where the optic nerve exits the back of the eye, is the result of cerebral vasodilation and elevated intracranial pressure. Table 2-11 illustrates some neurologic changes in hypercapnia and hypoxemia.

TABLE 2-11 Neurologic Changes in Hypercapnia and Hypoxemia

Condition	Physiologic Changes
Hypercapnia (with normal pH)	Increased cerebral blood flow Increased intracranial pressure
Hypercapnia (with low pH)	Impaired cerebral metabolism
Hypoxemia	Decreased mental and motor functions

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SUMMARY

Positive pressure ventilation is beneficial to support a patient's ventilatory and oxygenation needs. However, it has many inherent physiologic side effects on organ systems. When caring for critically ill patients with positive pressure ventilation, it is vital to observe and monitor the patients carefully and frequently for occurrence of side effects. Once the side effects are noted, appropriate interventions must be taken to correct the problems.

Self-Assessment Questions

1. During the inspiratory phase of positive pressure ventilation, gas flows into the lung because the pressure in the:

   A. airway is higher than the pressure in the lungs.
   B. airway is lower than the pressure in the lungs.
   C. lungs is higher than the barometric pressure.
   D. lungs and airway are the same.

2. A neonate is being ventilated by a pressure-controlled mode. The physician asks the therapist to adjust the ventilator to increase the patient's mechanical tidal volume. The therapist should:

   A. increase the tidal volume setting.
   B. increase the pressure setting.
   C. decrease the flow rate.
   D. decrease the inspiratory time.

3. A postoperative patient is recovering in the intensive care unit on volume-controlled mode. With this mode of ventilation, the ___ is preset with a variable ___ depending on the compliance and airway resistance characteristics.

   A. peak inspiratory pressure, tidal volume
   B. peak inspiratory pressure, peak flow
   C. tidal volume, peak inspiratory pressure
   D. tidal volume, peak flow

4. Positive pressure ventilation decreases the oxygen delivery to a patient with normal hemodynamic status because it causes all of the following changes *except*:

   A. increase in intrathoracic pressure.
   B. reduction in stroke volume.
   C. increase in pulmonary blood flow.
   D. decrease in cardiac output.

5. In children with right ventricular dysfunction, the thoracic pump mechanism ___ the outflow of blood from the ___ ventricle.

   A. increases, left
   B. decreases, left
   C. increases, right
   D. decreases, right

6 to 11. For *positive pressure ventilation,* match the volume and pressure changes with the respective reasons.

Pressure or Volume Changes	Reasons
6. *Increase* in intrathoracic pressure	A. Decreased venous return to right ventricle leads to lower right ventricular output.
7. *Decrease* in venous return or central venous pressure	B. Decreased right ventricular stroke volume and pulmonary artery pressure lead to lower left ventricular input and output.
8. *Decrease* in right ventricular stroke volume	C. Higher intrathoracic pressure impedes systemic blood return to right ventricle.
9. *Decrease* in pulmonary arterial pressure	D. Positive pressure applied to the lungs causes compression against the chest wall.
10. *Decrease* in ventricular filling pressures	E. Decreased right ventricular stroke volume leads to lower blood volume (pressure) in the pulmonary arteries.
11. *Decrease* in left ventricular stroke volume	F. Lower blood volume entering and leaving the ventricles.

12 to 15. For *positive end-expiratory pressure,* match the pressure and cardiac output changes with the respective reasons. Some answers may be used *more than once.*

Pressure or Cardiac Output Changes	Reasons
12. *Increase* in pulmonary artery pressure	A. Increase in pulmonary artery pressure causes a higher right ventricular pressure and hinders the blood return from systemic circulation to right heart. This causes backup of blood flow and increase in pressure in the systemic venous circulation.
13. *Increase* in central venous pressure (CVP)	B. This is due to the significant increase in intrathoracic pressure and significant *decrease* in left and right ventricular stroke volumes.
14. *Decrease* in aortic pressure	C. PEEP and positive pressure applied to the lungs cause significant compression of pulmonary blood vessels.
15. *Decrease* in cardiac output	

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16. A patient in the renal dialysis unit has recently been placed on a ventilator because of ventilatory and oxygenation failure. In caring for this patient, which of the following laboratory results would indicate that the patient's renal functions are failing?

    A. blood urea nitrogen of 30 mg/dL
    B. creatinine of 1.0 mg/dL
    C. potassium of 3 mEq/L
    D. sodium of 140 mEq/L

17. For patients with renal ___ or failure, the drug concentration in the circulation is usually ___ than normal when clearance of those drugs is dependent on proper renal perfusion.

    A. hyperperfusion, higher
    B. hyperperfusion, lower
    C. hypoperfusion, higher
    D. hypoperfusion, lower

18. Perfusion to the liver is usually affected by a high level of ___ and hepatic failure may be present when the ___.

    A. positive pressure ventilation, bilirubin level is less than 50 mg/L
    B. positive pressure ventilation, albumin level is less than 20 mg/L
    C. positive end-expiratory pressure, bilirubin level is greater than 50 mg/L
    D. positive end-expiratory pressure, albumin level is greater than 20 mg/L

19. Cardiovascular, renal, and pulmonary dysfunction may occur in patients with an intra-abdominal pressure ___ when positive end-expiratory pressure ___ is used during mechanical ventilation.

    A. greater than 15 mm Hg, greater than 15 cm H_2O
    B. greater than 20 mm Hg, greater than 15 cm H_2O
    C. less than 15 mm Hg, greater than 15 cm H_2O
    D. greater than 20 mm Hg, less than 15 cm H_2O

20. Gastrointestinal (GI) complications during mechanical ventilation may be caused by all of the following conditions *except*:

    A. increased intrathoracic pressure.
    B. increased cardiac output.
    C. hypoperfusion of the GI viscera.
    D. use of opiates and sedatives.

21. For patients with increased work of breathing and CO_2 retention, the caloric intake should be ___ than normal and the source of nutrition should be ___ based so as to provide maximum calorie intake and minimum CO_2 production.

    A. higher, fat
    B. higher, dextrose
    C. lower, fat
    D. lower, dextrose

22. Hyperventilation is sometimes provided for patients with increased intracranial pressure. This is done because respiratory ___ can reduce the intracranial pressure by ___ the cerebral blood vessels.

    A. acidosis, dilating
    B. acidosis, constricting
    C. alkalosis, dilating
    D. alkalosis, constricting

23. Headache, drowsiness, and irritability are some signs of altered ___ status resulting from hypoxemia and hypercapnia.

    A. renal
    B. hepatic
    C. nutritional
    D. neurologic

Answers to Self-Assessment Questions

1. A.
2. B.
3. C.
4. C.
5. C.
6. D.
7. C.
8. A.
9. E.
10. F
11. B.
12. C.
13. A.
14. B.
15. B.
16. A.
17. C.
18. C.
19. B.
20. B.
21. A.
22. D.
23. D.

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Chapter 3

Classification of Mechanical Ventilators

Gary C. White

Outline

- Introduction
- Ventilator Classification
- Ventilatory Work
- Input Power
- Drive Mechanism
  - *Piston Drive Mechanism*
  - *Bellows Drive Mechanism*
  - *Microprocessor-Controlled Pneumatic Drive Mechanism*
- Control Circuit
  - *Mechanical*
  - *Pneumatic*
  - *Fluidics*
  - *Electronic*
- Control Variables
  - *Pressure Controller*
  - *Volume Controller*
  - *Flow Controller*
  - *Time Controller*
- Phase Variables
  - *Trigger Variable*
  - *Limit Variable*
  - *Cycle Variable*
  - *Baseline Variable*
  - *Conditional Variable*
- Terminology of Ventilation Modes
  - *Volume-Controlled Ventilation*
  - *Pressure-Controlled Ventilation*
  - *Intermittent Mandatory Ventilation (IMV)*
  - *Pressure Support*
  - *Dual Control within a Breath*
  - *Dual Control Breath-to-Breath*
  - *Pressure-Limited Time-Cycled Breaths*
  - *Pressure-Limited Flow-Cycled Breaths*
  - *Automode*
  - *Proportional Assist Ventilation*
  - *Automatic Tube Compensation*
  - *Airway Pressure Release Ventilation*
- Output Waveforms
  - *Pressure Waveforms*
  - *Volume Waveforms*
  - *Flow Waveforms*

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Alarm Systems
  Input Power Alarms
  Control Circuit Alarms
  Output Alarms
Summary
Self-Assessment Questions
Answers to Self-Assessment Questions
References
Additional Resources

Key Terms

compressors
controller
cycle variable
flow-triggered
microprocessor
pneumatic drive mechanism
pressure-triggered
servo
sine wave
solenoid valves
time-triggered

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- List the 3 drive mechanisms of a ventilator.
- List and describe the 4 control circuits of a ventilator.
- Describe the 4 control variables (controllers) of a ventilator.
- Describe the 5 phase variables (controllers) of a ventilator.
- Explain the difference between volume-controlled ventilation and pressure-controlled ventilation.
- Name and describe the characteristics of 12 modes of ventilation.
- Identify the components of pressure, volume, and flow waveforms.
- Name the 3 alarm systems of a ventilator.

INTRODUCTION

The role of patient management in many situations is dependent on the respiratory care practitioner's understanding of mechanical ventilators and their characteristics. This applies to the acute care setting for all ages: adults, pediatrics, and neonatal patients. The same is also true in the transport and long-term care environments. Therefore, it is important for the respiratory care practitioner to understand classification of mechanical ventilators and the design characteristics employed by a ventilator to achieve the task of supporting a patient's ventilation.

In this chapter, you learn how ventilators are classified according to Chatburn's classification system and how the common ventilators employed in various patient care settings are classified.

VENTILATOR CLASSIFICATION

Ventilator technology has evolved since the introduction of Engström 100, the first volume-controlled mechanical ventilator in 1951. Since that time, a multitude of manufacturers have produced and marketed ventilators of all sizes, descriptions, and capabilities. Many manufacturers have coined new terms to describe their ventilators and to accentuate how their product is different from the others. Several different ventilator classification systems may be employed to describe mechanical ventilators. The majority of these systems focus on the differences between ventilators rather than the similarities.

Robert Chatburn (1992, 2007) has proposed a new way to classify mechanical ventilators based on related features, physics, and engineering. Chatburn's ventilator classification system has been featured in several articles and textbooks. It allows flexibility as ventilator technology evolves in contrast to other systems that employ more narrowly defined design principles or rely to a greater extent on manufacturer's terms.

With the evolution of ventilator technology over the next decade or more, the flexibility of Chatburn's classification system will be validated, as it is increasingly adopted by practitioners. This author believes this system is important enough to include in this text and in others that describe ventilator operational characteristics. Students and practitioners learning about this classification system should refer to the References section at the end of this chapter and read Chatburn's original contributions (Chatburn, 1991, 1992, 2001, 2007).

Ventilatory Work

Pulmonary physiologists have described the work ventilatory muscles perform during inspiration, and how muscles can actively assist during exhalation. During inspiration, the primary ventilatory muscles cause the size (volume) of the thoracic cage to increase, overcoming the elastic forces of the lungs and thorax and the resistance of the airways. As the volume of the thoracic cage increases, intrapleural pressure becomes more negative, resulting in lung expansion, as the visceral pleura expands with the parietal pleura. Gas flows from the atmosphere into the lungs as a result of the transairway pressure gradient. During expiration, the muscles of inspiration relax. The elastic forces of the lung and thorax cause the chest to decrease in volume. Exhalation occurs as a result of the greater pressure at the alveolus when compared to atmospheric pressure. All of this muscle activity to overcome the elastic and resistance properties of the lungs and thorax requires energy and work.

The work that the muscles and/or the ventilator must perform is proportional to the pressure required for inspiration times the tidal volume. The pressure required to deliver the tidal volume is referred to as the *load* either the muscles or the ventilator must work against. There is an elastic load (proportional to volume and inversely proportional to compliance) and a resistance load (proportional to airway resistance

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and inspiratory flow). These variables are related by the equation of motion for the respiratory system:

$$\text{Muscle Pressure} + \text{Ventilator Presssure} = \frac{\text{Volume}}{\text{Compliance}} + (\text{Resistance} \times \text{Flow})$$

Compliance is defined as a change in volume divided by a change in pressure, which is a measure of the elastic forces of the lungs and thorax. Flow, as defined earlier, is a unit of volume divided by a unit of time. Resistance is the force that must be overcome to move gas through the conducting airways, which is best described by Poiseuille's Law.

A mechanical ventilator is simply a machine or device that can fully or partially substitute for the ventilatory work accomplished by the patient's muscles. If the patient's ventilatory muscles contribute no work (sedation, paralysis, etc.), the mechanical ventilator provides full ventilatory support. If the patient's muscles are able to sustain all of the patient's ventilatory requirements, no support is provided by the machine and ventilatory support is zero. Between the two extremes, partial support can be provided by the mechanical ventilator in assisting the ventilatory muscles.

INPUT POWER

Mechanical ventilators may be first classified as to the power source that is used to provide the energy required to support the patient's ventilation. As described earlier, ventilation requires work and therefore, energy.

Pneumatically powered ventilators use compressed gas as an energy source for their operation. Medical gases are anhydrous (without water), and oil-free at a pressure of 50 psi. Examples of ventilators that utilize pneumatic power include the Bird Mark 7, Percussionaire IPV, Monaghan 225/SIMV, and the Percussionaire VDR.

Ventilators may also be electrically powered, utilizing 120 V 60 Hz alternating current (AC) or 12 V direct current (DC) for a power source. The electrical power can be used to run electric motors to drive pistons, **compressors**, or other mechanical devices that generate gas flow. Examples of electrically powered ventilators include the CareFusion LTV 1150 and Puritan Bennett 540.

compressors: A device capable of building up pressure by compressing the volume of air.

Some ventilators are powered by a combination of both pneumatic and electric power sources. Many third-generation ventilators require both an electrical (for **microprocessor**-controlled systems) and pneumatic power source. These ventilators include the Viasys AVEA, Puritan-Bennett 840, Hamilton-C2, among others.

microprocessor: Minute computer that is designed to perform specific functions.

DRIVE MECHANISM

The drive mechanism is the system used by the ventilator to transmit or convert the input power to useful ventilatory work. The type of drive mechanism determines the characteristic flow and pressure patterns each ventilator produces. The use of

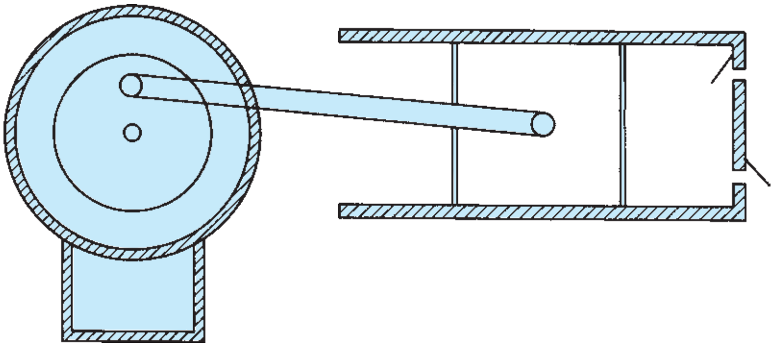

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FIGURE 3-1 A schematic diagram of a rotary-driven piston drive mechanism for a mechanical ventilator.

solenoid valves: A valve controlled by an electronic switching device that is used to regulate the specific functions of a ventilator.

microprocessors and proportional **solenoid valves** allow these newer ventilators to produce a variety of user-selected inspiratory flow or pressure patterns. An understanding of the different drive mechanisms will allow you to apply a ventilator more effectively in the clinical environment. Primary drive mechanisms include pistons, bellows, and pneumatic circuits.

Piston Drive Mechanism

An electrically driven piston with an inspiratory one-way valve can be used to generate a pressure gradient to drive a ventilator (Figure 3-1). During the backstroke of the piston, gas enters the cylinder through the one-way valve. When the piston travels in the opposite direction, a second one-way valve opens, delivering the compressed gas to the patient.

Pistons are usually electrically powered. However, they may be rotary- or linear-driven. Figure 3-2 compares a linear-driven and rotary-driven piston. Output

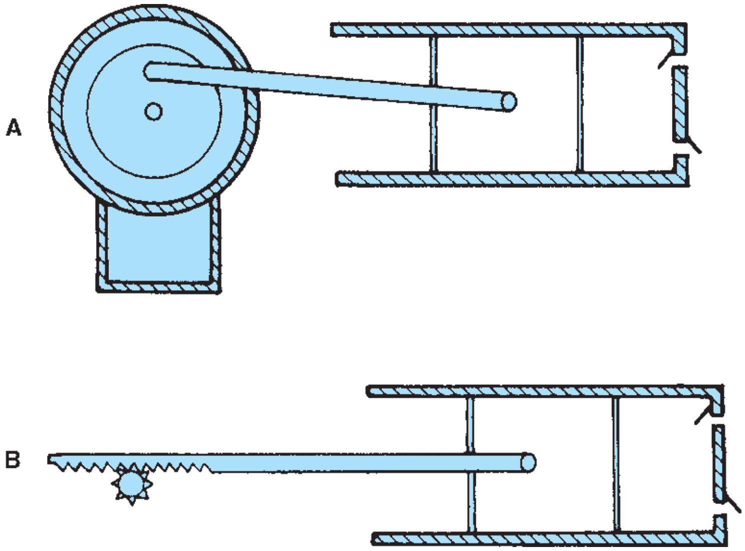

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FIGURE 3-2 A comparison between (A) a rotary-driven piston and (B) a linear-driven piston drive mechanism for a mechanical ventilator.

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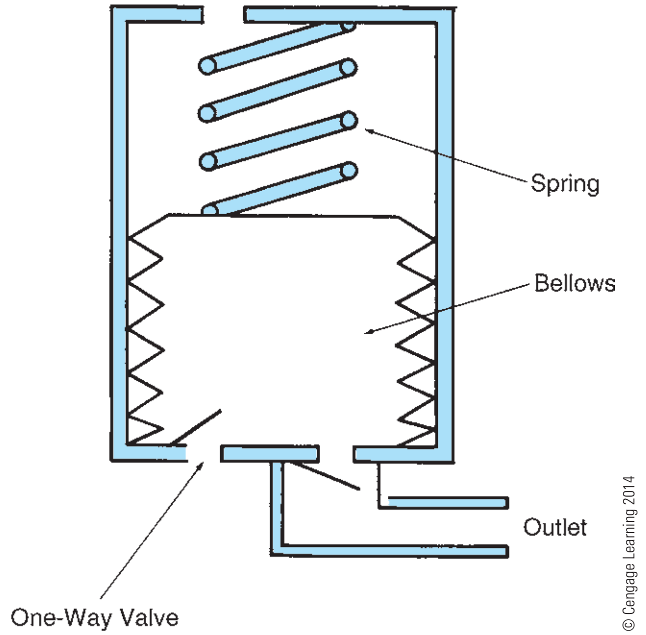

FIGURE 3-3 A bellows drive mechanism for a mechanical ventilator.

waveforms, which are discussed later in this chapter, vary depending on how the piston is driven.

Bellows Drive Mechanism

Ventilators may also use a bellows to compress the gas for delivery to the patient (Figure 3-3). A bellows may be compressed by a spring, a weight, or by gas pressure if it is in a sealed chamber. A one-way valve admits gas to the bellows expanding the bellows. When it is compressed, the one-way valve closes, causing gas delivery to the patient.

Microprocessor-Controlled Pneumatic Drive Mechanism

Although technically both pistons and bellows are pneumatic systems, a separate classification is required for the newer ventilators that use proportional solenoid valves and microprocessor controls. Current generation ventilators use programmed algorithms in the microprocessors to open and close the solenoid valves to mimic virtually any flow or pressure wave pattern.

This is called a microprocessor-controlled **pneumatic drive mechanism**. Furthermore, with advances in clinical medicine, the microprocessors can be reprogrammed to deliver new patterns that may not yet be described in the literature. Ventilator manufacturers, using microprocessors and the associated proportional solenoid valves, have greater flexibility in designing and updating ventilator technology.

pneumatic drive mechanism: Operation of a ventilator with pressurized gas as a power source.

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CONTROL CIRCUIT

The control circuit is the system that governs or controls the ventilator drive mechanism or output control valve. The control circuit is the system that is responsible for the characteristic output waveforms, which will be discussed later in this chapter. Control circuits may be classified as open- or closed-loop control circuits, mechanical, pneumatic, fluidics, and electronic.

An open-loop control circuit is one where the desired output is selected and the ventilator achieves the desired output without any further input from the clinician or the ventilator itself.

A closed-loop control circuit is one where the desired output is selected and then the ventilator measures a specific parameter or variable (flow, pressure, or volume) continuously, and the input is constantly adjusted to match the desired output. This type of control circuit may also be referred to as **servo**-controlled.

servo: A feedback system that typically consists of a sensing element, an amplifier, and a servomotor, used in the automatic control of the mechanical device of a ventilator.

Mechanical

Mechanical control circuits employ simple machines such as levers, pulleys, or cams to control the drive mechanism. Early mechanical ventilators used these systems to control their outputs. Being mechanical, some of these control systems were very durable but lacked flexibility by being an open-loop type control system.

Pneumatic

Pneumatic devices can be used as control circuits. These devices include valves, nozzles, ducted ejectors, and diaphragms. The IPPB ventilators and the Percussionaire IPV and VDR ventilators all use pneumatic control circuits.

Fluidics

Fluidics is the application of gas flow and pressure to control the direction of gas flows and to perform logic functions. The logic functions of fluidics have their origin in digital electronics. Fluidic elements, just as do digital electronic gates, control their outputs according to the inputs received. By combining fluidic elements in specific ways, a fluidic ventilator (e.g. Sechrist IV 100B) can be designed to function in a similar way to other ventilators that are electronically controlled.

Fluidic elements operate using the Coanda effect. If a jet of gas exits at high velocity adjacent to a wall (Figure 3-4), the gas flow will attach to the adjacent wall. An area of reduced pressure forms a separation bubble, which attaches the flow to the adjacent surface. Fluidic elements use a flow splitter located beside adjacent walls to control the direction of flow and to perform logic functions (Figure 3-5).

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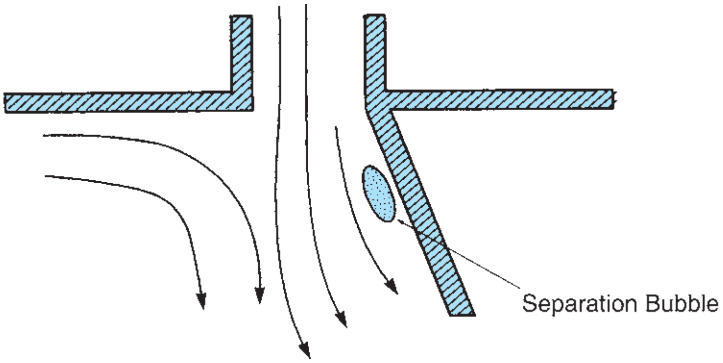

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FIGURE 3-4 A schematic illustrating the Coanda effect.

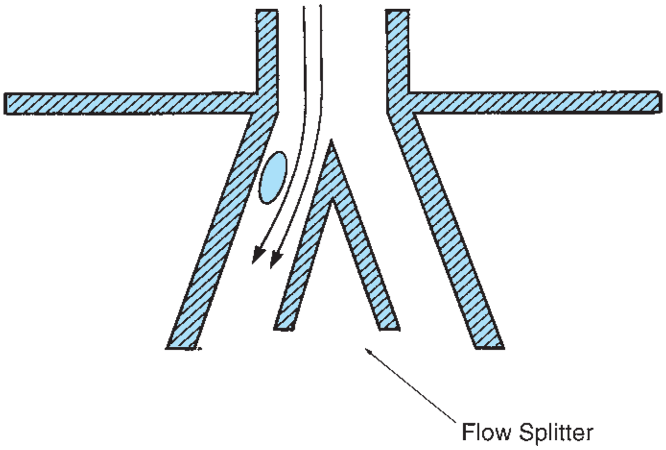

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FIGURE 3-5 A schematic illustrating a fluidic flow splitter.

Electronic

Electronic devices such as resistors, diodes, transistors, integrated circuits, and microprocessors can be used to provide sophisticated levels of control over the drive mechanisms of contemporary ventilators. Electronic control systems provide greater flexibility but often at the expense of complexity.

CONTROL VARIABLES

When providing ventilatory support, the mechanical ventilator can control four primary variables during inspiration. These four variables are pressure, volume, flow, and time. Figure 3-6 illustrates an algorithm that can be applied to determine which variable the ventilator is controlling.

controller: The mechanism that provides a mode of ventilation within a specific parameter (pressure, time, volume, or flow).

Pressure Controller

A ventilator is classified as a pressure **controller** if the ventilator controls the transrespiratory system pressure (airway pressure minus body surface pressure). Further

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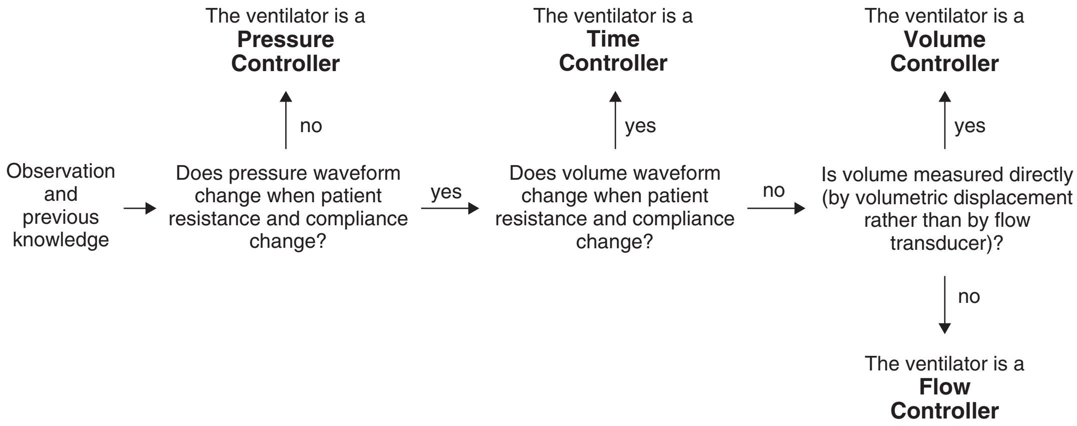
The ventilator is a **Pressure Controller**
no
The ventilator is a **Time Controller**
yes
The ventilator is a **Volume Controller**
yes
Observation and previous knowledge
Does pressure waveform change when patient resistance and compliance change?
yes
Does volume waveform change when patient resistance and compliance change?
no
Is volume measured directly (by volumetric displacement rather than by flow transducer)?
no
The ventilator is a **Flow Controller**

From R. L. Chatburn (1991). Respir Care, 36(10). Used with permission.

FIGURE 3-6 Criteria for determining the control variable during a ventilator-assisted inspiration.

classification of a ventilator as a positive or negative pressure ventilator depends on whether the airway pressure rises above baseline (positive) or body surface pressure is lowered below baseline (negative).

A positive pressure ventilator applies pressure inside the chest to expand it. This type of ventilator requires the use of a tight-fitting mask, or more commonly, an artificial airway. A pressure greater than atmospheric pressure is applied to the lungs, causing them to expand (Figure 3-7). Once positive pressure is no longer applied, the patient is allowed to exhale passively to ambient pressure. Exhalation occurs because of the pressure differential between the lungs and the atmosphere and through the elastic recoil of the lungs and thorax. This is the type of ventilator most commonly used today.

Negative pressure ventilators apply subatmospheric pressure outside of the chest to inflate the lungs. The negative pressure causes the chest wall to expand, and the pressure difference between the lungs and the atmosphere causes air to flow into the lungs (Figure 3-8). Once negative pressure is no longer applied, the patient is allowed to exhale passively to ambient pressure. Positive pressure may also be applied to further assist the patient during exhalation.

Regardless of whether a ventilator is classified as positive or negative pressure, the lungs expand as a result of the positive transrespiratory system pressures generated. It is the transrespiratory pressure gradient that largely determines the depth or volume of inspiration. A typical pressure controller is unaffected by

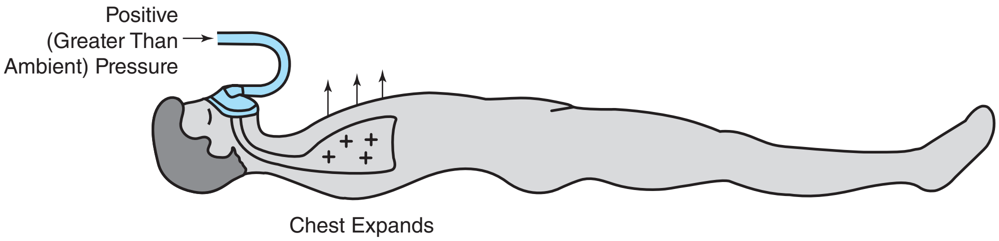
Positive (Greater Than Ambient) Pressure
+ +
+ +
Chest Expands

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FIGURE 3-7 A schematic illustrating positive pressure ventilation.

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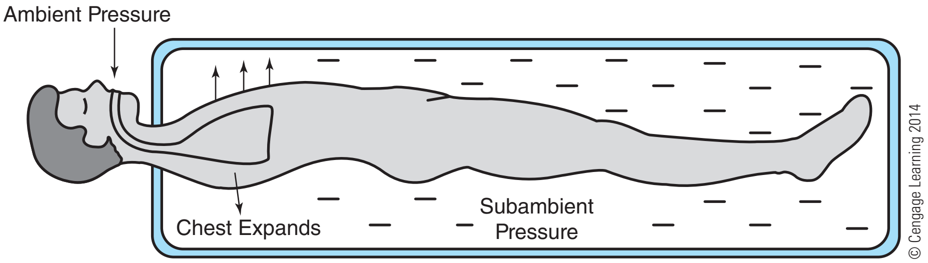

FIGURE 3-8 A schematic illustrating negative pressure ventilation.

changes in the patient's compliance or resistance. That is, the pressure level that is delivered to the patient will not vary in spite of changes in patient compliance or resistance.

Volume Controller

To be classified as a volume controller, volume must be measured and used as a feedback signal to control the output (volume) delivered. A volume controller allows pressure to vary with changes in resistance and compliance while volume delivery remains constant.

Volume controllers can measure volume by the displacement of the piston or bellows that serves as the ventilator's drive mechanism. If the displacement of the bellows or piston is controlled, volume therefore is also controlled.

Flow Controller

Flow controllers allow pressure to vary with changes in the patient's compliance and resistance while directly measuring and controlling flow. Flow may be measured by vortex sensors, heated wire grids, venturi pneumotachometers, strain gauge flow sensors, and other devices. What is important is that the ventilator directly measures flow and uses the flow signal as a feedback signal to control its output.

Many ventilators are incorrectly classified as *volume ventilators.* Even though a tidal volume is set or displayed, many ventilators measure flow and then derive volume from the flow measurement [Volume (L) = Flow (L/sec) × Inspiratory Time (sec)]. However, if a ventilator is operated in pressure support or pressure-controlled mode, the ventilator then becomes a pressure controller, since pressure is the variable that is measured and controlled.

Time Controller

Time controllers are ventilators that measure and control inspiratory and expiratory time. These ventilators allow pressure and volume to vary with changes in pulmonary compliance and resistance. Since neither pressure nor volume is directly measured or used as a control signal, time (inspiratory, expiratory, or both) remains the only variable that may be controlled.

PHASE VARIABLES

A ventilator-supported breath may be divided into four distinct phases: (1) the change from expiration to inspiration, (2) inspiration, (3) the change from inspiration to expiration, and (4) expiration. More detail can be learned by studying what occurs to the four variables (pressure, volume, flow, and time) during these phases. When the variable is examined during a particular phase, it is termed a *phase variable.*

Trigger Variable

The trigger variable is the variable that determines the start of inspiration. Pressure, volume, flow, or time may be measured by the ventilator and used as a variable to initiate inspiration. Many ventilators may use time or pressure as trigger variables.

time-triggered: Initiation of a mechanical breath based on the set time interval for one complete respiratory cycle (inspiratory time and expiratory time).

Control: Time-Triggered. A **time-triggered** breath is initiated and delivered by the ventilator when a preset time interval has elapsed. The frequency control on the ventilator is a time-triggering mechanism.

For example, if the ventilator frequency is preset at 12 breaths per minute (60 sec), the time-triggering interval for each complete breath is 5 sec. At this time-trigger interval, the ventilator automatically delivers one mechanical breath every 5 sec without regard to the patient's breathing effort or requirement.

$$60 \text{ sec}/12 \text{ breaths} = 5 \text{ sec/breath}$$

pressure-triggered: Initiation of a mechanical breath based on the drop in airway pressure that occurs at the beginning of a spontaneous inspiratory effort.

Pressure-Triggered. A **pressure-triggered** breath is initiated and delivered by the ventilator when it senses the patient's spontaneous (negative pressure) inspiratory effort. The patient may trigger the ventilator by generating a pressure gradient or a flow gradient.

Pressure triggering uses the drop in airway pressure that occurs at the beginning of a spontaneous inspiratory effort to signal the ventilator to begin inspiration (Figure 3-9). The amount of negative pressure, below the patient's baseline airway pressure (or end-expiratory pressure), that a patient must generate to trigger the ventilator into inspiration, is the sensitivity level. The range of acceptable sensitivity levels for pressure triggering varies from −1 to −5 cm H_2O below the patient's baseline pressure.

✓ Comparing to a sensitivity setting of −3 cm H_2O, −5 cm H_2O requires *more* patient effort to trigger the ventilator to inspiration.

For example, if the sensitivity for pressure triggering is set at −3 cm H_2O, then the patient must generate a pressure of −3 cm H_2O at the airway opening to trigger the ventilator into inspiration. If the sensitivity for pressure triggering is changed from −3 to −5 cm H_2O, the ventilator becomes less sensitive to the patient's inspiratory effort as more effort is needed to trigger the ventilator into inspiration. Changing the sensitivity from −3 to −5 cm H_2O is *decreasing* the sensitivity setting on the ventilator.

In situations where auto-PEEP is present, the triggering pressure would become greater. In order for the ventilator to sense a successful triggering effort, the patient must overcome both the auto-PEEP level and the sensitivity setting. See Figure 12-2

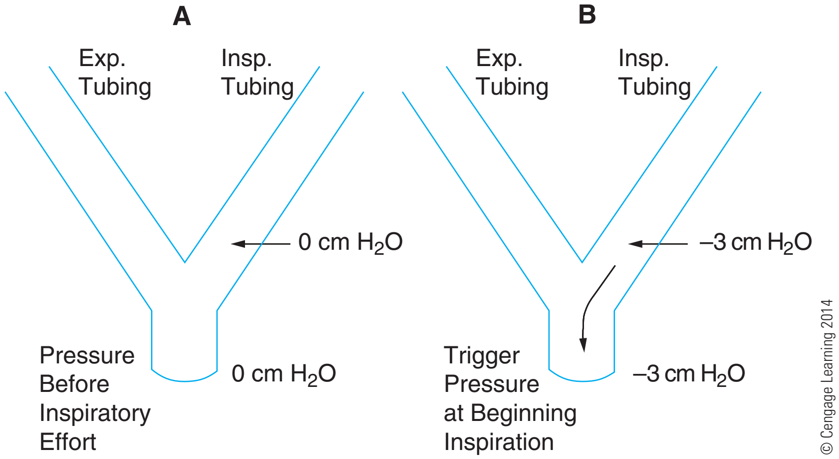

FIGURE 3-9 Pressure-trigger mechanism. (A) Before an inspiratory effort, the pressure in the airway and ventilator tubing equals 0 cm H_2O. A mechanical breath is not initiated because there is no pressure drop to trigger the ventilator sensitivity settings. (B) At beginning inspiration, the pressure in the airway and ventilator tubing is -3 cm H_2O. A mechanical breath is initiated because the pressure drop is sufficient to trigger the ventilator sensitivity setting (assuming it is set as -3 cm H_2O or less).

(Chapter 12) for further discussion on auto-PEEP and the strategy to compensate for the effects of auto-PEEP.

Flow-Triggered. Some ventilators are able to measure inspiratory and expiratory flows. When the patient's inspiratory flow reaches a specific value, a ventilator-supported breath is delivered. Flow triggering has been shown to be more sensitive and responsive to a patient's efforts than pressure triggering. A **flow-triggered** breath uses a strategy that combines the continuous flow and demand flow mechanisms, and it is used to reduce the inspiratory effort imposed on the patient during mechanical ventilation. It is considered to be more sensitive to the patient's inspiratory effort and therefore usually requires less inspiratory work than pressure triggering.

flow-triggered: Flow-triggering strategy uses a combination of continuous flow and demand flow. Before inspiration, the delivered flow equals the return flow. As the patient initiates a breath, the return flow to the ventilator is decreased and this flow differential triggers a mechanical breath.

In flow triggering, a continuous flow passes through the ventilator circuit and returns to the ventilator (i.e., delivered flow = returned flow). As the patient initiates a breath, part of the delivered flow goes to the patient and the return flow to the ventilator is therefore reduced (i.e., delivered flow > return flow). The ventilator senses this flow differential and instantly supplies enough flow to satisfy the mechanical or spontaneous tidal volume. CMV, SIMV, and PSV can all be flow-triggered (Figure 3-10).

How hard the patient must work to initiate or trigger a breath is termed the ventilator sensitivity. If the ventilator is made more sensitive to the patient's efforts (pressure, flow, or volume), it is easier for the patient to trigger a breath. The converse is also true.

Limit Variable

During a ventilator-supported breath, volume pressure and inspiratory flow all rise above their respective baseline values. Inspiratory time is defined as the time interval

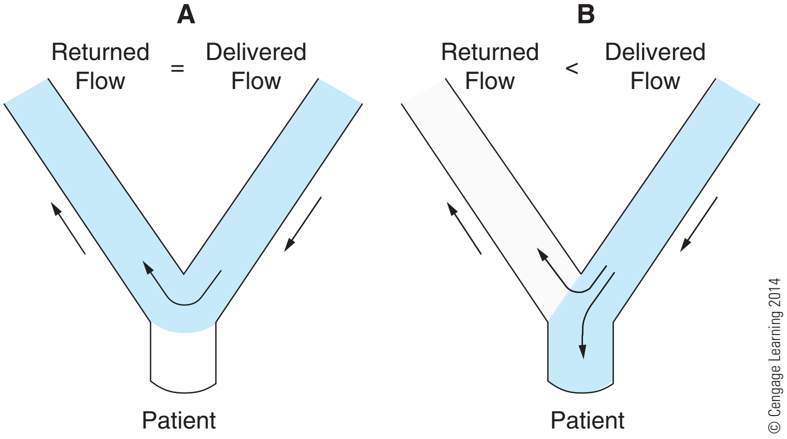

FIGURE 3-10 Flow-trigger mechanism. (A) Before an inspiratory effort, the delivered flow equals the returned flow. Flow trigger is not activated because there is no drop in returned flow. (B) At beginning inspiration, some of the delivered flow goes to the patient and this leads to a lower returned flow. Flow trigger is activated when the ventilator senses an inspiratory effort (the returned flow is lower than the delivered flow).

between the start of inspiratory flow and the beginning of expiratory flow. If one or more variables (pressure, flow, or volume) is not allowed to rise above a preset value during the inspiratory time, it is termed a limit variable. In this definition, inspiration does not end when the variable reaches its preset value. The breath delivery continues, but the variable is held at the fixed, preset value. (Note that the *pressure limit variable* discussed above is not the same as the *high pressure limit* one would set on the ventilator to prevent excessive pressure during inspiration.) Figure 3-11 provides a useful algorithm for determining the limit variable (pressure-limited, volume-limited, or flow-limited) during the inspiratory phase.

Cycle Variable

cycle variable: A measurement that causes the breath to end.

Inspiration ends when a specific **cycle variable** (pressure-cycled, volume-cycled, flow-cycled, or time-cycled) is reached (Figure 3-11). This variable must be measured by the ventilator and used as a feedback signal to end inspiratory flow delivery, which then allows exhalation to begin.

Again, it is easy to make false assumptions regarding many ventilators by classifying them as volume-cycled. Most newer ventilators measure flow and are flow controllers. Since flow is measured and used as a feedback signal for gas delivery, volume becomes a function of flow and time [Volume (L) = Flow (L/sec) × Inspiratory Time (sec)]. Therefore, these ventilators are really time-cycled, rather than "volume-cycled." Inspiration ends because a preset time interval has passed, and volume has not been directly measured.

Baseline Variable

Expiratory time is defined as the interval between the start of expiratory flow and the beginning of inspiratory flow of the next breath. This is also termed the

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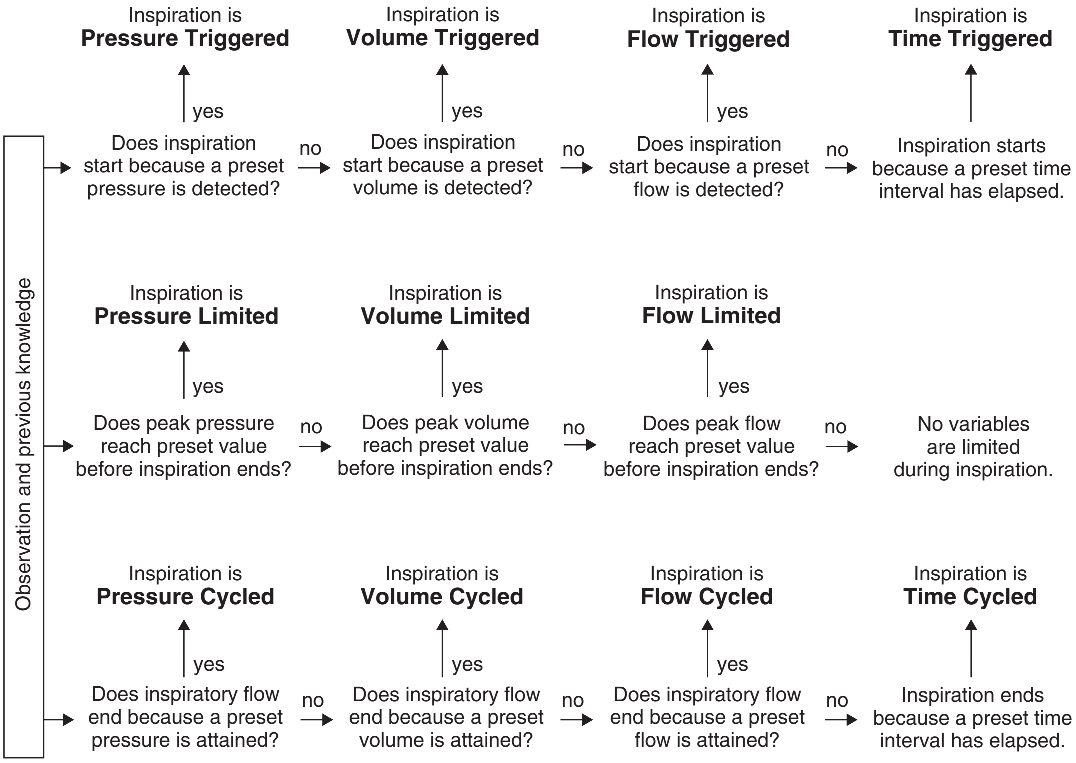
Inspiration is
Pressure Triggered
yes
Does inspiration start because a preset pressure is detected?
no
Inspiration is
Volume Triggered
yes
Does inspiration start because a preset volume is detected?
no
Inspiration is
Flow Triggered
yes
Does inspiration start because a preset flow is detected?
no
Inspiration is
Time Triggered
Inspiration starts because a preset time interval has elapsed.
Inspiration is
Pressure Limited
yes
Does peak pressure reach preset value before inspiration ends?
no
Inspiration is
Volume Limited
yes
Does peak volume reach preset value before inspiration ends?
no
Inspiration is
Flow Limited
yes
Does peak flow reach preset value before inspiration ends?
no
No variables are limited during inspiration.
Inspiration is
Pressure Cycled
yes
Does inspiratory flow end because a preset pressure is attained?
no
Inspiration is
Volume Cycled
yes
Does inspiratory flow end because a preset volume is attained?
no
Inspiration is
Flow Cycled
yes
Does inspiratory flow end because a preset flow is attained?
no
Inspiration is
Time Cycled
Inspiration ends because a preset time interval has elapsed.
Observation and previous knowledge

From R. L. Chatburn (1991). *Respir Care, 36*(10). Used with permission.

FIGURE 3-11 Criteria for determining the phase variables during a ventilator-assisted breath.

expiratory phase. The variable that is controlled during the expiratory phase or expiratory time is termed the baseline variable. Most commonly, pressure is controlled during the expiratory phase.

Application of positive end-expiratory pressure (PEEP) and continuous positive airway pressure (CPAP) are used to increase the functional residual capacity (FRC), to improve gas distribution, and oxygenation. These pressures, when applied above baseline (ambient pressure) during exhalation, maintain the lungs in a partially inflated state. This helps to prevent alveolar collapse, inflate previously collapsed alveoli, and distend those alveoli that are already patent. PEEP and CPAP pressures must be titrated carefully to monitor hemodynamic functions, blood gases or oximetry, and compliance, to achieve the greatest benefit with the least amount of detrimental side effects.

Conditional Variable

Conditional variables are changes detected by the ventilator when a certain threshold is met, resulting in a designated output. Early ventilators, such as the Puritan Bennett MA-1, used relatively simple conditional variables (volume-cycled, pressure-limited, pressure-triggered, and PEEP). Newer third-generation microprocessor-controlled ventilators, such as the Puritan-Bennett 840, are capable of delivering complex ventilatory patterns. Figure 3-12 summarizes the ventilator classification system.

	Mandatory Breath				Spontaneous Breath				Control Logic		
Mode	**Control**	**Trigger**	**Limit**	**Cycle**	**Control**	**Trigger**	**Limit**	**Cycle**	**Supported**	**Conditional Variable**	**Action**
CMV	Pressure, volume, or flow	Time	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	—	—
A/C	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	Time or patient effort	Machine-to-patient triggered
AMV	Pressure, volume, or flow	Pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	—	—
IMV	Pressure, volume, or flow	Time	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	—	—
SIMV	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
CPAP	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	—	—
PCV	Pressure	Time	Pressure	Time	—	—	—	—	—	—	—
PC-IMV	Pressure	Time	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No		

FIGURE 3-12 Summary of the ventilator classification system as described by Robert L. Chatburn.

	Mandatory Breath				Spontaneous Breath				Control Logic		
Mode	Control	Trigger	Limit	Cycle	Control	Trigger	Limit	Cycle	Supported	Conditional Variable	Action
PC-SIMV	Pressure	Time, pressure, volume, or flow	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
PCIRV	Pressure	Time	Pressure	Time	Pressure						
APRV	Pressure	Time or pressure	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
PSV	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Volume	Yes		
MMV	Volume or flow	Time	Volume or flow	Time, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure or volume	Yes	Minute ventilation, time	Spontaneous-to-mandatory breath
VAPS	Flow	Time or pressure	Flow	Time or volume	Pressure	Pressure or flow	Pressure	Flow	Yes	Tidal volume	Pressure-to-volume control
BiPAP	Pressure	Time	Pressure	Time	Pressure	Pressure	Pressure	Pressure	No	—	—

CMV = continuous mandatory ventilation; NA = not applicable; A/C = assist/control; AMV = assisted mechanical ventilation; IMV = intermittent mandatory ventilation; SIMV = synchronized mandatory ventilation; CPAP = continuous positive airway pressure; PCV = pressure-controlled ventilation; PC-IMV = pressure-controlled IMV; PCIRV = PC inverse-ratio ventilation; APRV = airway pressure release ventilation; PSV = pressure support ventilation; MMV = mandatory minute ventilation; VAPS = volume-assisted pressure support; BiPAP = bilevel positive airway pressure.

FIGURE 3-12 *(continued)*

From R. L. Chatburn (1992), "Technical description and classification of modes of ventilator operation," Respir Care, 37(9). Used with permission.

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TERMINOLOGY OF VENTILATION MODES

A mechanical ventilation mode is defined as “a specific combination of breathing pattern, control type, and operational algorithms” (Chatburn, 2007). With the advent of microprocessor-controlled ventilators, the variety and complexity of modes has dramatically increased (Branson et al., 2004). It is important to understand mechanical ventilation modes in order to match breath delivery to specific clinical application and patient needs. This section provides the terminology of basic ventilation modes. Chapter 4 will discuss the clinical application of commonly used modes of ventilation along with the respective waveforms.

Volume-Controlled Ventilation

Volume-controlled ventilation allows the clinician to set the volume to delivered with each breath (Campbell et al., 2002). With volume delivery fixed, pressure will vary, depending upon the patient’s pulmonary compliance and airway resistance (Figure 3-13). Volume will remain constant in spite of changes in the patient’s condition. The advantage of volume control is the ability to regulate both tidal volume and minute ventilation (a product of tidal volume and frequency).

Pressure-Controlled Ventilation

The pressure-controlled mode allows the clinician to set a peak inspiratory pressure for each mechanical breath (Campbell et al., 2002). Since pressure remains constant, volume and minute ventilation will vary with changes in the patient’s pulmonary compliance or airway resistance (Figure 3-14). Should the patient’s compliance worsen or airway resistance increase, the peak inspiratory pressure terminates soon and the tidal volume and minute ventilation decreases. The advantage of the pressure-controlled mode is that the lungs can be protected from excessive pressures, preventing ventilator-induced lung injury (VILI).

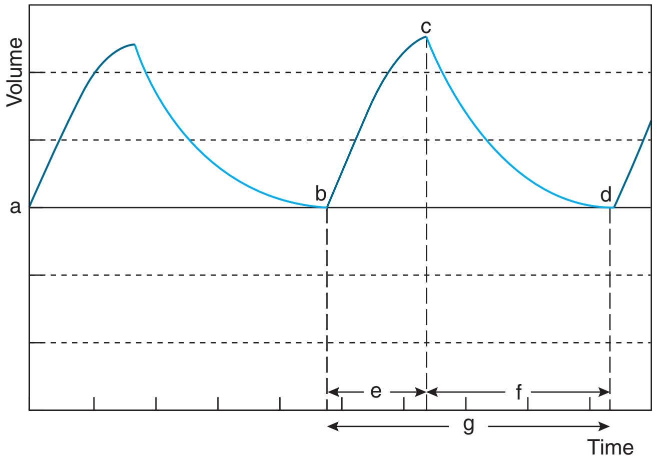

FIGURE 3-13 A volume-time scalar in pressure-controlled mode. a: beginning inspiration, b: end-expiration/beginning inspiration, c: end-inspiration/beginning expiration, d: end-expiration, e: inspiratory time, f: expiratory time, g: total cycle time.

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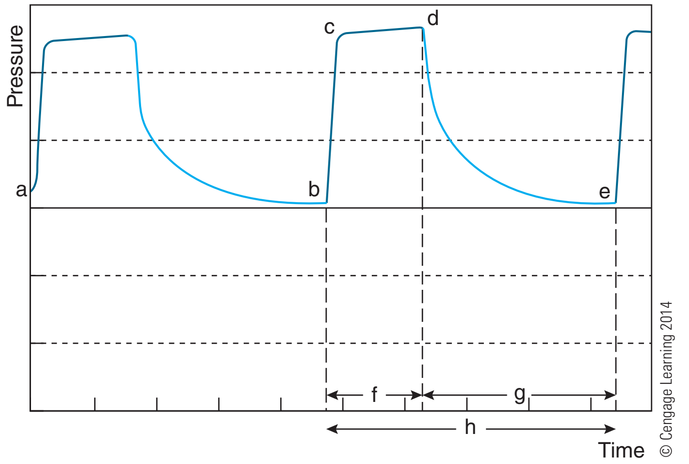

FIGURE 3-14 A pressure-time scalar in pressure-controlled mode. A pressure-time scalar in pressure-controlled mode. a: beginning inspiration, b: end-expiration/beginning inspiration, c to d: pressure plateau, d: end-inspiration/beginning expiration, e: end-expiration, f: inspiratory time, g: expiratory time, h: total cycle time.

Intermittent Mandatory Ventilation (IMV)

Intermittent mandatory ventilation (IMV) allows the patient to spontaneously breathe between time-triggered ventilator breaths, which may be volume- or pressure-controlled. The patient is able to breathe gas at the same F_IO_2 and baseline pressure, without triggering a mandatory breath (ventilator breath). Spontaneous breaths may also be augmented using pressure support to increase the patient's spontaneous tidal volume and to reduce some of the inspiratory work associated with the endotracheal tube's resistance. Figure 3-15 illustrates the IMV mode.

Pressure Support

Pressure support is a variation of the spontaneous mode of ventilation that augments a patient's spontaneous effort with positive pressure. It is a spontaneous ventilation mode in which the patient must trigger each breath (pressure- or flow-triggered). This mode augments spontaneous ventilation, increasing tidal volume with the application of adjustable pressure. On initiation of a breath, a constant pressure (preset) is delivered until the flow rate reaches between 10% to 40% of the peak inspiratory flow; then, expiration begins. In this mode, flow is variable, and flow will increase to a level needed to maintain the desired pressure support level. This mode assures that the patient's spontaneous breaths are large enough to maintain adequate blood

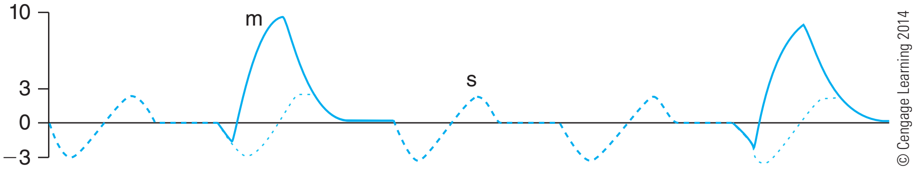

FIGURE 3-15 A scalar presentation of intermittent mandatory ventilation (IMV). m = mechanical breath; s = spontaneous breath

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gases and to prevent atelectasis. The patient demand and pathology (resistance and compliance) determines the volume delivered and the spontaneous frequency.

Dual Control within a Breath

Dual control within a breath implies that two variables become control variables during inspiration within the same breath. During dual control within-a-breath modes, the ventilator switches from pressure-controlled to volume-controlled. The clinician sets a desired tidal volume, which becomes a volume target during the breath. The ventilator begins the breath as a pressure controller, delivering a constant pressure initially during the breath. During breath delivery, tidal volume is measured and the pressure is adjusted automatically by the ventilator to maintain the guaranteed tidal volume (volume control). Dual control within-a-breath modes establish a high initial inspiratory flow (pressure-controlled breath), and a taper or plateau in flow as the volume target is met. Examples of this mode include pressure augmentation and volume-assured pressure support.

Dual Control Breath-to-Breath

Dual control breath-to-breath modes allow the clinician to set a volume target, and the ventilator delivers pressure-controlled breaths attempting to achieve the desired target tidal volume. The ventilator may operate in either pressure support or pressure-controlled mode, with the pressure limit increasing or decreasing to achieve the desired volume target (Branson et al., 2004).

Pressure-Limited Time-Cycled Breaths

Pressure-limited time-cycled breaths begin inspiration as pressure-limited breaths (pressure increases to a set value or target), and they are time-cycled (inspiration ends at a specified time interval). The clinician sets a target tidal volume and maximum pressure (pressure limit). The ventilator delivers a test breath and calculates the patient's airway resistance and lung compliance. Once resistance and compliance have been determined, pressure increases or decreases automatically to reach the desired volume target. Pressure is adjusted in increments of 1 to 3 cm H_2O at a time between breaths, until the maximum pressure is reached or a set level below the upper pressure limit. If the desired volume is not met, an alarm alerts the clinician to the fact and the upper pressure limit is never exceeded. Examples of dual control breath-to-breath pressure-limited, time-cycled modes include volume control plus (VC+) and pressure-regulated volume control (PRVC).

Pressure-Limited Flow-Cycled Breaths

Pressure-limited flow-cycled breaths start as a pressure-support breath with a target tidal volume. Inspiration is flow-cycled (inspiration ends when inspiratory flow falls to predetermined value). The clinician sets a volume target, PEEP and pressure limit. The breath begins as a pressure-controlled (pressure-support) breath and the ventilator measures the tidal volume delivered. If the tidal volume falls below the target level, inspiratory pressure is increased on the next breath to attempt to achieve the target. As in pressure support, the breath ends when inspiratory flow decays to a percentage of the peak flow. If the patient becomes apneic, the ventilator switches to a volume-controlled backup ventilation mode. This mode is available on the Maquet Servo-i as Volume Support Ventilation (VSV).

Automode

Automode combines PRVC and volume support into a single mode (Branson et al., 2004). In the absence of spontaneous patient effort, the ventilator delivers mandatory breaths using a time-triggered, pressure-limited, time-cycled mode, adjusting the pressure limit to maintain the clinician-set tidal volume. If the patient breathes spontaneously for two consecutive breaths, the ventilator will switch to volume-support ventilation (VSV) where the breaths are patient-triggered (pressure or flow), pressure-limited, and flow-cycled. If the patient becomes apneic (12 seconds for adults, 8 seconds for pediatrics), the ventilator will switch back to PRVC mode.

Proportional Assist Ventilation

Proportional assist ventilation (PAV) is a mode in which the ventilator will proportionally assist the patient's spontaneous ventilation. The ventilator does so by proportionally amplifying the delivered pressure (pressure support) in proportion to the measured inspiratory flow and volume (Branson et al., 2004). The amount of support provided by the ventilator is tailored or adjusted to the patient's spontaneous effort, increasing or decreasing pressure support relative to the patient's work of breathing. PAV may be pressure- or flow-triggered and is cycled when the patient's volume or flow demands are met. PAV is available on the Puritan-Bennett 840 ventilator.

Automatic Tube Compensation

Automatic tube compensation is a mode of ventilation that automatically compensates for the resistance of the endotracheal tube. The pressure applied is based upon the size and type of artificial airway (endotracheal tube or tracheostomy tube) and how much support is desired by the clinician. Automatic tube compensation can eliminate the resistance imposed by the artificial airway. The ventilator adjusts the pressure to compensate for airway size or flow demands (Branson et al., 2004). Automatic tube compensation is active both during inspiration and expiration, and may reduce air trapping and intrinsic PEEP. Automatic tube compensation is available on the Puritan-Bennett 840 ventilator.

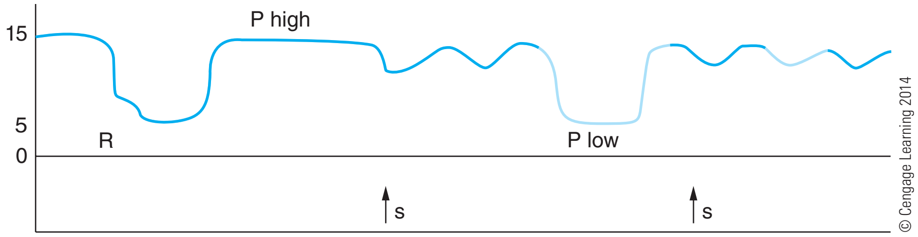


FIGURE 3-16 A scalar presentation of airway pressure release ventilation (APRV) mode.
A scalar presentation of airway pressure release ventilation (APRV) mode.
P high. Upper (higher) airway pressure (15 cm H_2O)
P low. Lower airway pressure (PEEP of 5 cm H_2O as shown)
R. Pressure release 10 cm H_2O (15 cm H_2O - 5 cm H_2O)
S. Beginning of spontaneous breaths at P high

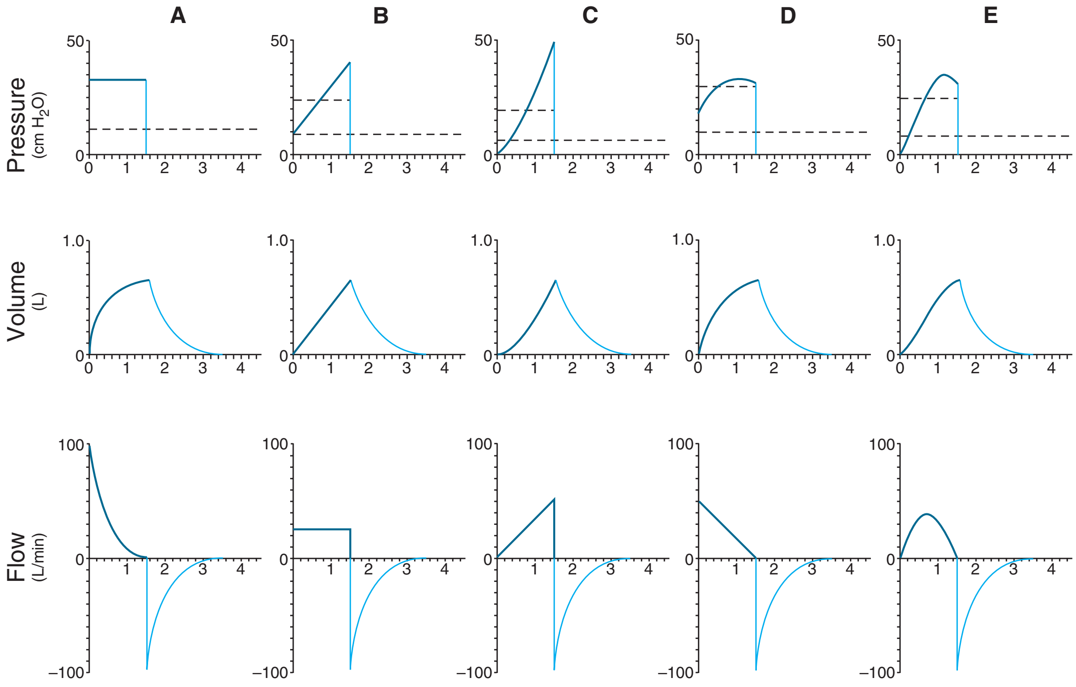
Airway Pressure Release Ventilation

Airway pressure release ventilation (APRV) is a form of continuous positive airway pressure (CPAP) with two distinct pressure levels. APRV maintains spontaneous breathing throughout the entire ventilatory cycle at both pressure levels (Figure 3-16). APRV is a time-triggered, pressure-limited, and time-cycled mode which allows spontaneous breathing.

The clinician sets the high and low pressures, and the inspiratory times at each pressure level. Typically, the higher pressure is initially set above the lower inflection point of the lung's pressure volume curve, close to what the mean airway pressure would be during pressure-controlled ventilation (Myers, 2007). The higher pressure keeps the alveoli inflated and enhances recruitment. The time interval at the higher pressure (T_{high}) is longer than the time spent at the lower pressure (T_{low}). Release of the pressure from the higher to the lower pressure setting helps to facilitate removal of CO_2 (Myers, 2007). Time triggering is established using set time intervals for T_{high} and T_{low}. Additionally, most ventilators allow patient triggering of a breath (pressure or flow). Some manufacturers also permit the application of pressure support during the spontaneous portion at the higher CPAP level.

OUTPUT WAVEFORMS

Output waveforms are graphical representations of the control or phase variables in relation to time. Output waveforms are typically presented in the order of pressure, volume, and flow. The ventilator determines the shape of the control variable, whereas the other two depend on the patient's compliance and resistance. Convention dictates that flow values above the horizontal axis are inspiratory, whereas flow below the horizontal axis is expiratory. This corresponds to pressure



From R. L. Chatburn (1991). *Respir Care, 36*(10). Used with permission.

FIGURE 3-17 Theoretical output waveforms for (A) pressure-controlled inspiration with rectangular pressure waveform, identical to flow-controlled inspiration with an exponential-decay flow waveform; (B) flow-controlled inspiration with rectangular flow waveform, identical to volume-controlled inspiration with an ascending-ramp flow waveform; (C) flow-controlled inspiration with an ascending-ramp flow waveform; (D) flow-controlled inspiration with a descending-ramp flow waveform; and (E) flow-controlled inspiration with a sinusoidal flow waveform. The short dashed lines represent mean inspiration pressure, whereas the longer dashed lines denote mean airway pressure (assuming zero end-expiratory pressure). For the rectangular pressure waveform in A, the mean inspiratory pressure is the same as the peak inspiratory pressure. These output waveforms were created by (1) defining the control waveform (e.g., an ascending-ramp flow waveform is specified as flow = constant 3 time) and specifying that tidal volume equals 644 mL (about 9 mL/kg for a normal adult); (2) specifying the desired values for resistance and compliance (for these waveforms, compliance = 20 mL/cm H_2O and resistance = 20 cm H_2O/L/sec, according to ANSI recommendations); (3) substituting the above information into the equation of motion; and (4) using a computer to solve the equation for pressure, volume, and flow and plotting the results against time.

and flow values rising above the horizontal axis for inspiration and falling back to the baseline during expiration. The ideal waveforms are represented in Figure 3-17.

Careful observation and assessment of waveforms during mechanical ventilation can provide useful information for the clinician. Waveforms can assist the clinician in the detection of inadvertent PEEP, the patient's ventilatory work, resistance and compliance changes, as well as many other events or changes. Some ventilators are able to present pressure versus volume waveforms to assist in minimizing the patient's work of breathing. Still other ventilators can present flow versus volume waveforms, to aid in the assessment of airway obstruction and the effectiveness of bronchodilator therapy during mechanical ventilation. As waveforms become widely used, their usefulness will approach that of the ECG tracing in the assessment of the heart.

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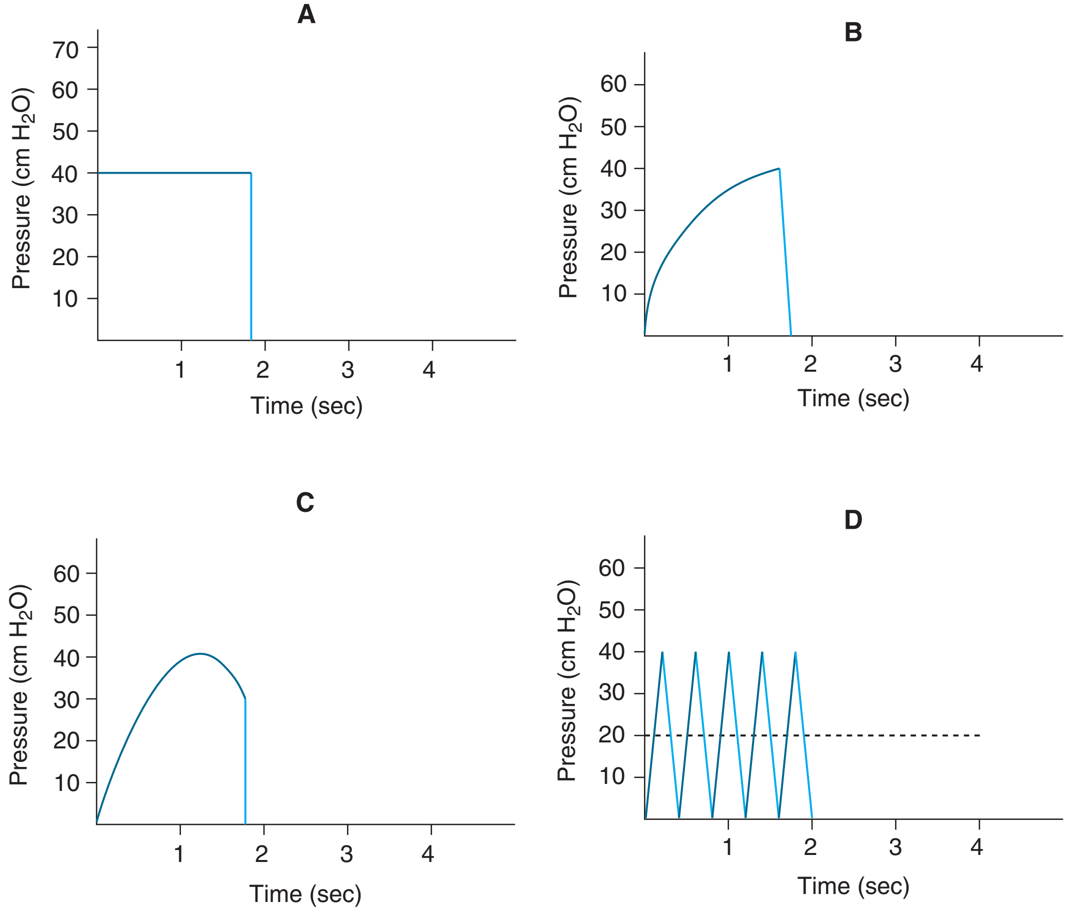

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FIGURE 3-18 Four types of pressure waveforms: (A) rectangular; (B) exponential; (C) sinusoidal; and (D) oscillating.

Pressure Waveforms

Pressure waveforms include rectangular, exponential, sinusoidal, and oscillating (Figure 3-18). Each of these waveforms would have these characteristic shapes, providing that pressure is the control variable. The descriptors used to describe each waveform are based on their respective shapes.

The rectangular waveform is characterized by a nearly instantaneous rise to a peak pressure value that is held to the start of exhalation. During expiration, the pressure rapidly drops to baseline.

The exponential waveform is depicted by a more gradual increase in pressure when compared with the rectangular waveform. This type of waveform is common in some infant ventilators and has become an option on some adult ventilators. Ventilator settings such as flow and inspiratory time regulate how steep the waveform rises toward peak inspiratory pressure.

sine wave: A graphic presentation of flow and time that has a horizontal "S" appearance.

The sinusoidal waveform resembles the positive half of a **sine wave**. Sinusoidal waveforms are characteristically produced by ventilators having a rotary-driven

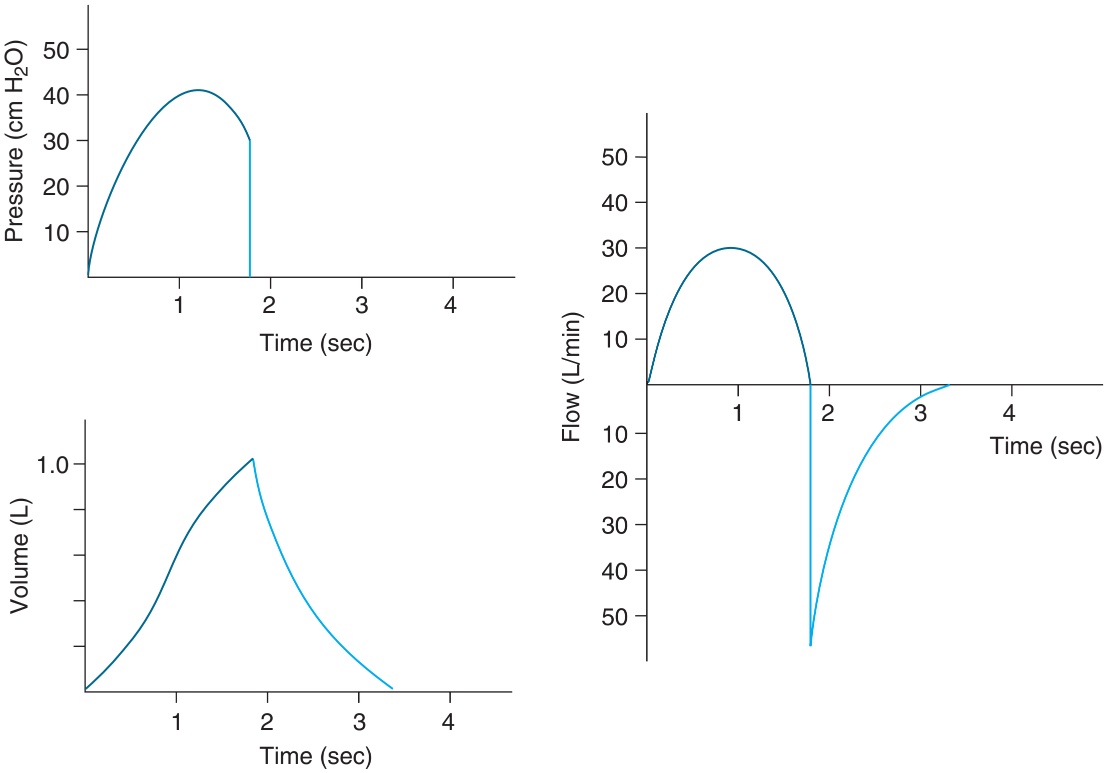

FIGURE 3-19 Sinusoidal pressure waveform illustrated with the corresponding volume and flow waveforms. This type of pattern is typical of a rotary-driven piston drive mechanism.

piston drive mechanism (Figure 3-19). Ventilators using this drive mechanism include the Emerson 3-MV, Respironics PLV-100, Bear 33, and Puritan-Bennett LP-10 and LP-20.

Volume Waveforms

Volume waveforms can be classified into two types, ascending ramp and sinusoidal (Figure 3-20). The ascending ramp waveform is produced by a constant (i.e.,

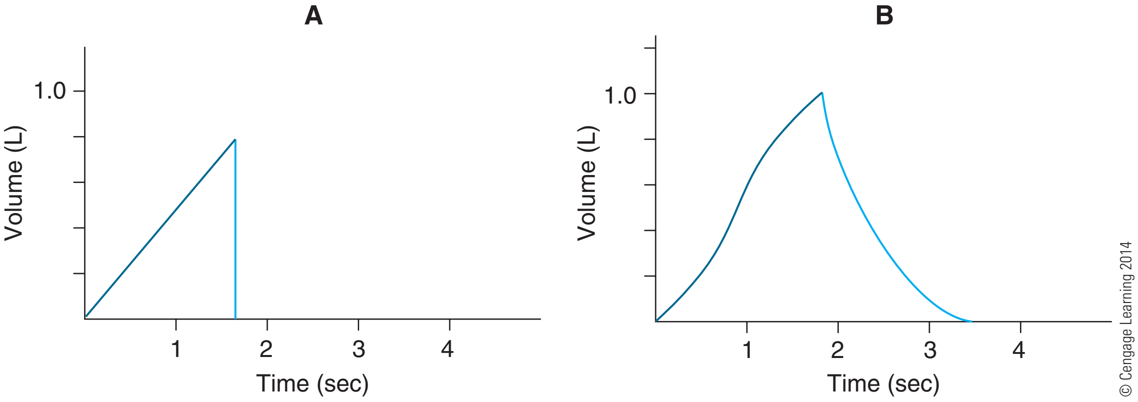

FIGURE 3-20 Two types of volume waveforms: (A) ascending ramp; and (B) sinusoidal.

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rectangular) inspiratory flow pattern. The shape is characterized by a linear rise to the peak inspiratory pressure value. Sinusoidal volume waveforms are produced by ventilators that have a rotary-driven piston drive mechanism. Ventilators using this drive mechanism include the Emerson 3-MV, Respironics PLV-100, BEAR 33, and Puritan-Bennett LP-10 and LP-20.

Flow Waveforms

The four types of flow waveforms are shown in Figure 3-21. The waveforms include rectangular (constant), ascending ramp, descending ramp, and sinusoidal.

The rectangular waveform is produced when volume is the control variable and the output is an ascending ramp. The flow waveform (a derivative of the

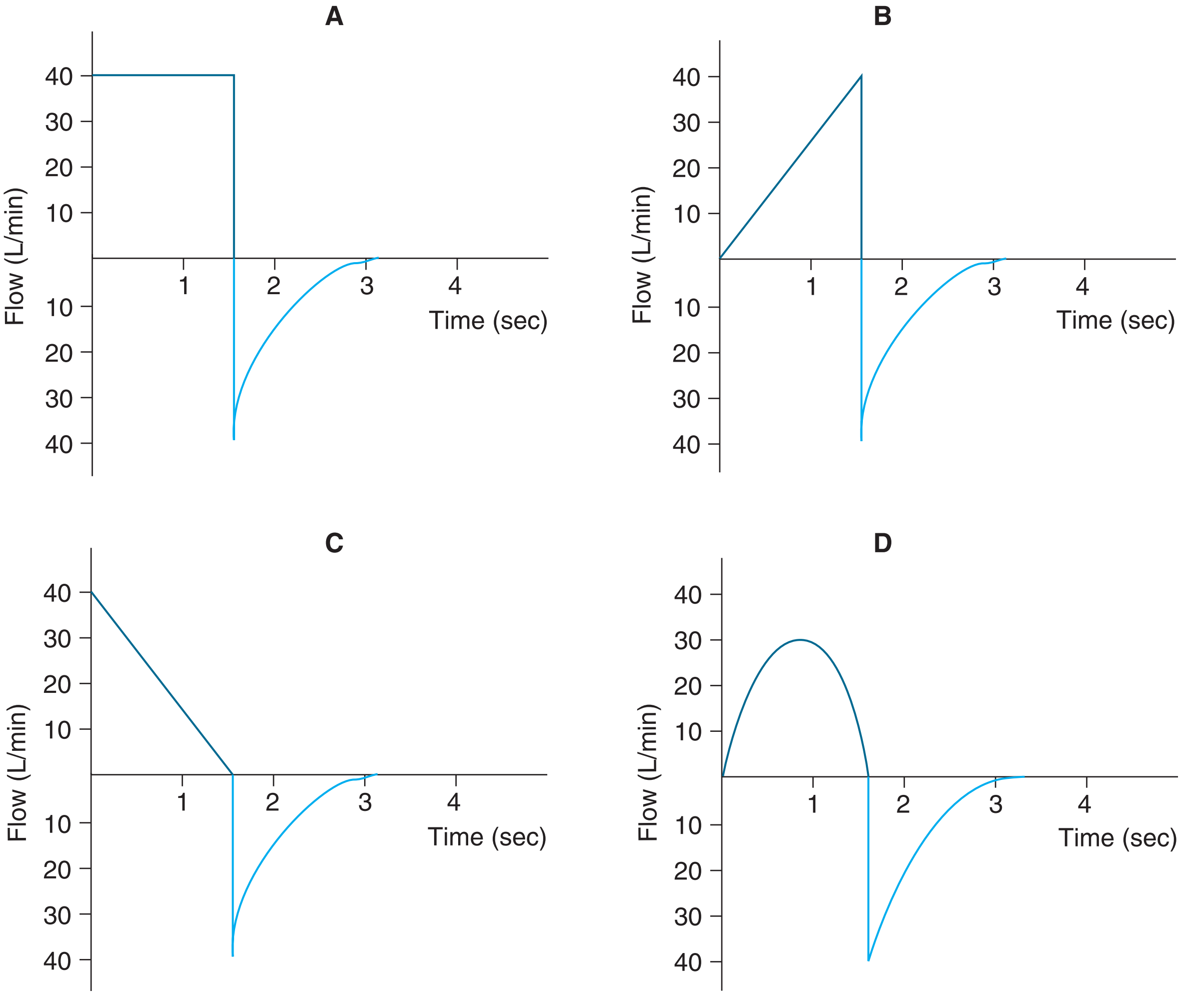

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FIGURE 3-21 Four types of flow waveforms: (A) rectangular (constant); (B) ascending ramp; (C) descending ramp; and (D) sinusoidal.

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volume waveform with respect to time) assumes the characteristic rectangular shape.

The ramp waveform can be ascending or descending. If flow rises as the breath is delivered, it is termed ascending. If flow falls during the ventilator-supported breath, it is called a descending ramp.

The sinusoidal waveform resembles the positive portion of a sine wave. It is generated by a rotary-driven piston drive mechanism.

ALARM SYSTEMS

Alarm systems are designed to alert the clinician to undesirable technical or patient events. Triggering of any alarm requires clinician awareness or action. As the complexity of mechanical ventilators has increased, so have the number and complexity of the alarm systems. Technical events are those events limited to the performance of the ventilator, while patient events are those relating to the patient's condition. Alarms can be visual, audible, or both, depending on the seriousness of the event.

Input Power Alarms

Input power alarms can be further classified as to loss of electrical or pneumatic power.

Loss of electrical power usually results in the ventilator activating a backup alarm that is battery-powered. Most battery backup alarms are powered by rechargeable nickel cadmium batteries, which are recharged when AC power is available. When AC power is lost, the backup batteries activate audible and visual alarms.

Loss of either air or oxygen pneumatic sources will result in a technical event alarm. If either input pressure falls below a specified value from 50 psi, the alarm will result. Some alarms are electronic (BEAR 1000, BEAR I, II and III, Puritan-Bennett 840), whereas others are pneumatic reed alarms, such as those employed in oxygen blenders.

Control Circuit Alarms

Control circuit alarms alert the clinician to settings or parameters that are not within acceptable ranges or specifications, or they warn the clinician that the ventilator has failed some part of a self-diagnostic test. In the event of an incompatible setting or parameter, the clinician is allowed the opportunity to change the input to one that is compatible. Failure of the self-diagnostic test may render the ventilator inoperative, and the clinician is alerted by a message display that test failure has occurred.

Output Alarms

Output alarms can be further subdivided into pressure, volume, flow, time, inspiratory, and expiratory gas.

Pressure alarms include high/low peak and mean and baseline airway pressures. High and low values may be set for each of these output parameters to alert the clinician of changes in the patient's respiratory status. Additionally, an alarm may be provided to detect failure of the airway pressure to return to the baseline valve. This could be caused by airway obstructions, circuit obstructions, or ventilator malfunctions.

Volume alarms include high/low exhaled tidal volumes for both ventilator-supported breaths and spontaneous breaths. Low volumes may result from sedation (spontaneous volumes), disconnection, or apnea (spontaneous volumes).

Flow alarms are limited to exhaled minute volume. High and low values may be set on some ventilators to alert the clinician to changes in the patient's minute ventilation.

Time alarms include high/low frequency, excessive or inadequate inspiratory or expiratory time and inverse I:E ratio. High/low frequency alarms alert the clinician to changes in the total ventilatory frequency. Inspiratory and expiratory time alarms may alert the practitioner to circuit obstructions or malfunctions, changes in gas distribution, or inappropriate ventilator settings.

Inspired gas alarms alert the clinician to changes in oxygen concentration or gas temperature. Some ventilators incorporate an oxygen analyzer to detect changes in F_IO_2. High/low alarms alert the clinician to these changes. Inspired gas temperature may be controlled by a servo-controlled humidifier or monitored by an independent ventilator temperature alarm. High/low temperature alarms can alert the clinician to changes in the inspiratory gas temperature.

Exhaled oxygen tension or end tidal carbon dioxide tension can be monitored, and high/low alarms can be sent to the exhaled gas monitoring system. These monitors can assist the clinician in determining the V_D/V_T, gas exchange, and the respiratory exchange ratio (R).

SUMMARY

As computer and medical technologies are getting more advanced, future mechanical ventilators are likely to have more new features than the current ventilators. No one knows for certain whether more new features will make the ventilators more complex and less user-friendly. But no matter what the future ventilators become, the practitioners who use mechanical ventilators must learn and maintain the theory, skills, and practice in the use of mechanical ventilation.

The ability to use mechanical ventilation will be enhanced if the practitioners are able to classify the ventilator properly and apply the unique characteristics of each ventilator in patient care situations.

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Self-Assessment Questions

1. The primary forces that the ventilatory muscles must overcome include:

    I. resistive forces
    II. compliance forces
    III. elastic forces
    IV. inductive forces

    A. I and II.
    B. I and III.
    C. II and III.
    D. II and IV.

2. When the ventilator assumes all of the ventilatory work, this is termed:

    A. partial ventilatory support.
    B. full ventilatory support.
    C. incomplete ventilatory support.
    D. no ventilatory support.

3. A ventilator that measures flow and uses that measurement to control the output of the ventilator is termed a:

    A. pressure controller.
    B. flow controller.
    C. volume controller.
    D. time controller.

4. When the ventilator delivers a mechanical breath in response to the patient's inspiratory effort, the mechanical breath is called:

    A. time-triggered.
    B. volume-triggered.
    C. flow-triggered.
    D. pressure- or flow-triggered.

5. If pressure rises to a preset level and is maintained at that level until inspiration ends, this is termed a:

    A. time limit.
    B. volume limit.
    C. pressure limit.
    D. flow limit.

6. An alarm that results from the loss of 50 psi gas pressure is termed a(n):

    A. input power alarm.
    B. control circuit alarm.
    C. output alarm.
    D. gas pressure alarm.

7. PEEP and CPAP are modes in which the pressure is set:

    A. at the baseline pressure.
    B. above the baseline pressure.
    C. below the baseline pressure.
    D. 5 to 10 cm H_2O above the baseline pressure.

8. In volume-controlled ventilation, the peak inspiratory pressure increases when the patient's:

    A. compliance or airway resistance is increased.
    B. compliance or airway resistance is decreased.
    C. compliance is increased or airway resistance is decreased.
    D. compliance is decreased or airway resistance is increased.

9. In pressure-controlled ventilation, the delivered volume is increased when the patient's:
    - A. compliance or airway resistance is increased.
    - B. compliance or airway resistance is decreased.
    - C. compliance is increased or airway resistance is decreased.
    - D. compliance is decreased or airway resistance is increased.

10. A mode of ventilation that augments a patient's spontaneous tidal volume with a preset level of pressure is called:
    - A. Pressure-controlled.
    - B. Pressure limit.
    - C. Pressure support.
    - D. Volume-controlled.

Answers to Self-Assessment Questions

1. B.
2. B.
3. B.
4. D.
5. C.
6. A.
7. B.
8. D.
9. C.
10. C.

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Chapter 4

Operating Modes of Mechanical Ventilation

David W. Chang
James H. Hiers

Outline

- Introduction
- Negative and Positive Pressure Ventilation
  - *Negative Pressure Ventilation*
  - *Positive Pressure Ventilation*
- Operating Modes of Mechanical Ventilation
- Closed-Loop System
- Spontaneous
  - *Apnea Ventilation*
- Positive End-Expiratory Pressure (PEEP)
  - *Indications for PEEP*
  - *Physiology of PEEP*
  - *Complications of PEEP*
- Continuous Positive Airway Pressure (CPAP)
- Bilevel Positive Airway Pressure (BiPAP)
  - *Indications for BiPAP*
  - *Initial Settings*
  - *Adjustments of IPAP and EPAP*
- Controlled Mandatory Ventilation (CMV)
  - *Indications for Control Mode*
  - *Complications of Control Mode*
- Assist/Control (AC)
  - *Assist Control Triggering Mechanism*
  - *Assist Control Cycling Mechanism*
  - *Indications for AC Mode*
  - *Advantages of AC Mode*
  - *Complications of AC Mode*
- Intermittent Mandatory Ventilation (IMV)
- Synchronized Intermittent Mandatory Ventilation (SIMV)
  - *SIMV Mandatory Breath-Triggering Mechanism*
  - *SIMV Spontaneous Breath-Triggering Mechanism*
  - *Indications for SIMV Mode*
  - *Advantages of SIMV Mode*
  - *Complications of SIMV Mode*

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- Mandatory Minute Ventilation (MMV)
- Pressure Support Ventilation (PSV)
  - *Indications for PSV Mode*
- Adaptive Support Ventilation (ASV)
- Proportional Assist Ventilation (PAV)
- Volume-Assured Pressure Support (VAPS)
- Pressure-Regulated Volume Control (PRVC)
  - *Automode*
- Adaptive Pressure Control (APC)
- Volume Ventilation Plus (VV+)
  - *Volume Control Plus (VC+)*
  - *Volume Support (VS)*
- Pressure-Controlled Ventilation (PCV)
- Airway Pressure Release Ventilation (APRV)
  - *Indications for APRV*
- Biphasic Positive Airway Pressure (Biphasic PAP)
- Inverse Ratio Ventilation (IRV)
  - *Physiology of IRV*
  - *Adverse Effects of IRV*
  - *Pressure Control-IRV (PC-IRV)*
- Automatic Tube Compensation (ATC)
- Neurally Adjusted Ventilatory Assist (NAVA)
- High Frequency Oscillatory Ventilation (HFOV)
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

- adaptive pressure control (APC)
- adaptive support ventilation (ASV)
- airway pressure release ventilation (APRV)
- assist/control (AC)
- automatic tube compensation (ATC)
- automode
- biphasic positive airway pressure (biphasic PAP)
- bilevel positive airway pressure (BiPAP)
- continuous positive airway pressure (CPAP)
- control mode
- eucapnic ventilation
- high frequency oscillatory ventilation (HFOV)
- intermittent mandatory ventilation (IMV)
- mandatory minute ventilation (MMV)
- neurally adjusted ventilatory assist (NAVA)
- positive end-expiratory pressure (PEEP)
- pressure-controlled ventilation (PCV)
- pressure-regulated volume control (PRVC)
- proportional assist ventilation (PAV)
- synchronized intermittent mandatory ventilation (SIMV)
- volume-assured pressure support (VAPS)
- volume ventilation plus (VV+)

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Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Differentiate negative pressure ventilation and positive pressure ventilation.
- Describe the characteristics and clinical application of the following terms or modes of ventilation: spontaneous, positive end-expiratory pressure, continuous positive airway pressure, bilevel positive airway pressure, controlled mandatory ventilation, assist/control, intermittent mandatory ventilation, synchronized intermittent mandatory ventilation, mandatory minute ventilation, and pressure support ventilation.
- Describe the characteristics and clinical application of the following terms or modes of ventilation: adaptive support ventilation, proportional assist ventilation, volume-assured pressure support, pressure-regulated volume control, volume ventilation, pressure-controlled ventilation, airway pressure release ventilation, biphasic positive airway pressure, inverse ratio ventilation, pressure-controlled inverse ratio ventilation, automatic tube compensation, and neurally adjusted ventilator assist.

INTRODUCTION

This chapter provides an introduction to different operating modes available on most mechanical ventilators. Since the information associated with mechanical ventilation is immense, the reader should learn the operating modes and study them in the order presented in this chapter. The definition and unique characteristics of each operating mode are described here. The initiation and application of these operating controls may be found in subsequent chapters.

NEGATIVE AND POSITIVE PRESSURE VENTILATION

✓ Mechanical ventilators generate gas flow and volume by creating either a negative or positive pressure gradient.

Every ventilator must generate an inspiratory flow in order to deliver a tidal volume. Since gas flow requires a pressure gradient, a mechanical ventilator must produce a pressure gradient (i.e., pressure difference) between the airway opening and the alveoli in order to produce inspiratory flow and volume delivery. The pressure gradient that must be generated between the airway opening and the alveoli is known as the transairway pressure (Des Jardins, 2001).

$$\text{Transairway Pressure } (P_{TA}) = \text{Pressure at Airway Opening } (P_{AO}) - \text{Alveolar Pressure } (P_{ALV})$$

At end-exhalation and prior to the beginning of inspiration, the pressures at the airway opening and the alveoli are both equal to atmospheric pressure. Since these two pressures are equal at this point, there is no pressure gradient and therefore no flow.

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Since a pressure gradient is needed to generate gas flow and volume, mechanical ventilators achieve this condition by creating either a negative or positive pressure gradient.

Negative Pressure Ventilation

Negative pressure ventilation creates a transairway pressure gradient by decreasing the alveolar pressures to a level below the airway opening pressure (i. e., below the atmospheric pressure). Unless airway obstruction is present, negative pressure ventilation does not require an artificial airway. Two classical devices that provide negative pressure ventilation are the "iron lung" and the chest cuirass or chest shell.

Iron Lungs. An "iron lung" ventilator encloses the patient's body except for the head and neck in a tank, and the air in it is evacuated to produce a negative pressure around the chest cage. This negative pressure surrounding the chest and underlying alveoli results in chest wall and alveolar expansion. The tidal volume delivered to the patient is directly related to the negative pressure gradient. For example, a more negative pressure applied to the chest wall will yield a larger tidal volume. Since negative pressure ventilation does not require tracheal intubation, this noninvasive method of ventilation has been used extensively and successfully to support chronic ventilatory failure (Corrado et al., 1994; Frederick, 1994).

✓ The tidal volume delivered by a negative pressure ventilator is directly related to the negative pressure gradient.

Disadvantages and complications associated with the iron lung type of negative pressure ventilator are (1) poor patient access and (2) potential for a decreased cardiac output known as "tank shock" (Frederick, 1994).

Since the iron lung encloses the patient, it restricts access to the patient for routine health care. Tank shock may result from a decreased venous blood return to the right atrium. Normally, the heart and vena cava are surrounded by negative pleural pressure, while the remainder of the vascular system outside the thorax is subjected to atmospheric pressure. This creates a vascular pressure gradient between the vena cava and the venous drainage that enhances venous blood return to the right atrium. However, if a patient is placed in an iron lung, this vascular pressure gradient is lost because the peripheral vasculature is subjected to negative pressures that closely approximate the pleural pressure. This results in a potential decrease in venous return that could lead to a decreased cardiac output.

Chest Cuirass. The chest cuirass or chest shell (see Figure 18-1) is a form of negative pressure ventilation that was intended to alleviate the problems of patient access and tank shock associated with iron lungs. This shell device covers only the patient's chest and leaves the arms and lower body exposed. Although the chest shell improves patient access and decreases the potential for tank shock, ventilation with this device may be limited by the difficulties in maintaining an airtight seal between the shell and the patient's chest wall (Newman et al., 1988).

To overcome the problem of air leakage, individually designed cuirass "respirators" minimize air leaks, and they have been used successfully to ventilate patients with chest wall diseases such as scoliosis (Kinnear et al., Hockley, 1988; Kinnear et al., Petch, 1988). Because of the availability of positive pressure ventilators, chest cuirass ventilators are seldom used in an acute care facility. However, they are rather useful in selected home care settings because of the ease to maintain and the capability to ventilate without an artificial airway. All subsequent discussions on mechanical ventilation in this text refer to positive pressure ventilation unless negative pressure ventilation is specifically mentioned.

Positive Pressure Ventilation

✓ The tidal volume delivered by a positive pressure ventilator is directly related to the positive pressure gradient.

Positive pressure ventilation is achieved by applying positive pressure (a pressure greater than atmospheric pressure) at the airway opening. Increasing the pressure at the airway opening produces a transairway pressure gradient that generates an inspiratory flow. This flow, in turn, results in the delivery of a tidal volume. Therefore, tidal volume is directly related to the transairway pressure gradient. All other factors being held constant, increasing the positive pressure being applied to the lungs will result in a larger tidal volume being delivered.

OPERATING MODES OF MECHANICAL VENTILATION

A ventilator mode can be defined as a set of operating characteristics that control how the ventilator functions. An operating mode can be described by the way a ventilator is triggered into inspiration and cycled into exhalation, what variables are limited during inspiration, and whether or not the mode allows mandatory breaths, intermittent breaths, or spontaneous breaths.

Many additional operating functions are also available on modern ventilators. Some examples are control of the F_IO_2, control of the inspiratory flow rate, and control of various alarms.

Regardless of which operating mode is selected, it should achieve four main goals: provide adequate ventilation and oxygenation, avoid ventilator-induced lung injury, provide patient-ventilator synchrony, and allow successful weaning from mechanical ventilation. Table 4-1 provides an overview of the mechanical breath terminology that should enhance an understanding in the selection and use of different operating modes (Mireles−Cabodevila et al., 2009). Common operating modes will be reviewed separately in this chapter.

There are at least 23 modes of ventilation available in different ventilators. Two or more of these modes are often used together to achieve certain desired effects. For example, spontaneous plus PEEP is the same as CPAP, and it is used to oxygenate a patient who has adequate spontaneous ventilation. SIMV may be used with PSV to provide mechanical ventilation and reduce the work of spontaneous breathing.

1. Spontaneous
2. Positive end-expiratory pressure (PEEP)
3. Continuous positive airway pressure (CPAP)
4. Bilevel positive airway pressure (BiPAP)
5. Controlled mandatory ventilation (CMV)
6. Assist/control (AC)

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Table 4-1 Overview of Mechanical Breath Terminology

1. Mechanical Breath Variables	
Control variable	Mechanism to deliver a breath (e.g., pressure-controlled or volume-controlled)
Trigger variable	Mechanism to start inspiration (e.g., pressure or flow trigger by patient, time trigger by ventilator)
Cycle variable	Mechanism to end inspiration (e.g., volume-cycled, pressure-cycled, flow-cycled, time-cycled)
2. Breath Sequence	
Continuous mandatory	All breaths are controlled by the ventilator. No spontaneous breaths are allowed (e.g., CMV)
Intermittent mandatory	Set number of mandatory breaths are provided by the ventilator. Spontaneous breaths are allowed between mandatory breaths (e.g., SIMV).
Continuous spontaneous	All breaths are spontaneous with assistance (e.g., pressure support ventilation or PSV) or without assistance (e.g., continuous positive airway pressure or CPAP).
3. Type of Control or Target Scheme	
Set point	How the ventilator reaches its targeted goal (e.g., set point for pressure-controlled ventilation is pressure)
Servo	How the ventilator adjusts its output to suit the patient's variable (e.g., proportional assist ventilation adjusts its pressure to generate an appropriate flow to meet the patient's flow demand)
Adaptive	How the ventilator adjusts a set point to reach a different targeted set point (e.g., pressure-regulated volume control adjusts the pressure (by altering the flow and I-time) to reach a targeted volume.
Optimal	How the ventilator uses a mathematical model to alter the set points to achieve a target goal (e.g., adaptive support ventilation alters the frequency, tidal volume, and pressure to achieve a target minute ventilation.

7. Intermittent mandatory ventilation (IMV)
8. Synchronized intermittent mandatory ventilation (SIMV)
9. Mandatory minute ventilation (MMV)
10. Pressure support ventilation (PSV)

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11. Adaptive support ventilation (ASV)
12. Proportional assist ventilation (PAV)
13. Volume-assured pressure support (VAPS)
14. Pressure-regulated volume control (PRVC)
15. Adaptive pressure control (APC)
16. Volume ventilation plus (VV+)
17. Pressure-controlled ventilation (PCV)
18. Airway pressure release ventilation (APRV)
19. Biphasic positive airway pressure (Biphasic PAP)
20. Inverse ratio ventilation (IRV)
21. Automatic tube compensation (ATC)
22. Neurally adjusted ventilator assist (NAVA)
23. High-frequency oscillatory Ventilation (HFOV)

CLOSED-LOOP SYSTEM

In mechanical ventilation, a simple operating mode provides one output based on one input. An example is the tidal volume in volume-controlled ventilation. The clinician sets the tidal volume (input), and the ventilator delivers the tidal volume using a constant flow (output). The set tidal volume (input) is constant, and inspiratory flow (output) does not vary during the delivery of the tidal volume breath.

In other operating modes, the output changes during delivery of a mechanical breath. Pressure support ventilation is an example. During pressure support ventilation, the clinician sets the peak pressure plateau (input) and the ventilator uses a variable flow (output) to maintain the pressure plateau. Since the variable flow (output) is dependent on the changing characteristics of the airways and lungs, pressure support ventilation is a closed-loop system in which the input (set pressure) is constant and the output (flow) is variable (Branson et al., 2002).

In addition to pressure support ventilation, other examples of a closed-loop system in mechanical ventilation include mandatory minute ventilation, adaptive support ventilation, proportional assist ventilation, volume-assured pressure support, pressure-regulated volume control, and neurally adjusted ventilator assist.

SPONTANEOUS

Spontaneous setting on the ventilator is not an actual mode since the frequency and tidal volume during spontaneous breathing are determined by the patient. The ventilator simply provides the flow and supplemental oxygen. Even though the spontaneous mode is not a direct ventilator function, the role of the ventilator during

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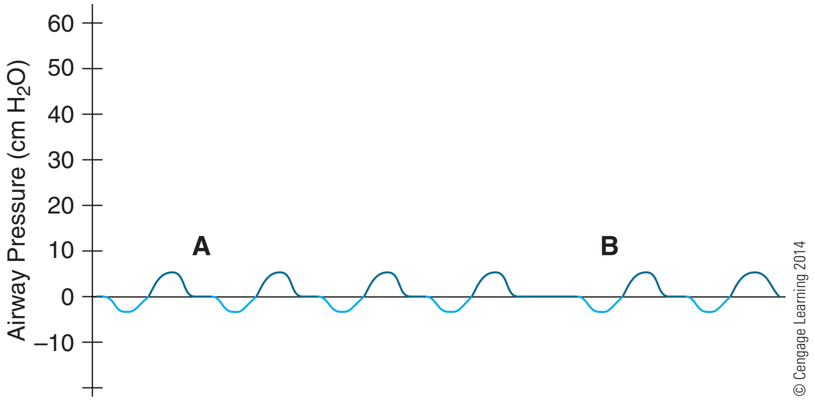

FIGURE 4-1 Spontaneous breathing pressure tracing. (A) The spontaneous rate is at a normal pattern. (B) The spontaneous breath is delayed by the patient.

the spontaneous mode is to provide the (1) inspiratory flow to the patient in a timely manner, (2) flow adequate to fulfill a patient's inspiratory demand (i.e., tidal volume or inspiratory flow), and (3) adjunctive modes such as PEEP to complement a patient's spontaneous breathing effort. The graphical tracing of spontaneous breaths is shown in Figure 4-1.

Apnea Ventilation

Apnea ventilation is a safety feature incorporated with the spontaneous breathing mode. In the event of apnea or an extremely low frequency, backup ventilation is invoked by the apnea ventilation feature and it delivers a predetermined tidal volume, frequency, F_IO_2, and other essential ventilator functions to the patient. Proper operation of apnea ventilation should be checked for each patient to ensure safety.

positive end-expiratory pressure (PEEP): PEEP is an airway pressure strategy in ventilation that increases the end-expiratory or baseline airway pressure to a value greater than atmospheric pressure. It is used to treat refractory hypoxemia caused by intrapulmonary shunting.

POSITIVE END-EXPIRATORY PRESSURE (PEEP)

Positive end-expiratory pressure (PEEP) increases the end-expiratory or baseline airway pressure to a value greater than atmospheric (0 cm H_2O on the ventilator manometer). It is often used to improve the patient's oxygenation status, especially in hypoxemia that is refractory to high level of F_IO_2.

PEEP is not commonly regarded as a "stand-alone" mode, rather it is applied in conjunction with other ventilator modes. For example, when PEEP is applied to spontaneous breathing patients, the airway pressure is called **continuous positive airway pressure (CPAP).** Figure 4-2 shows an **assist/control (AC)** pressure tracing with 10 cm H_2O of PEEP.

continuous positive airway pressure (CPAP): CPAP is PEEP applied to the airway of a patient who is breathing spontaneously. It is used to treat refractory hypoxemia in patients who are able to maintain adequate spontaneous ventilation.

assist/control (AC): In the assist/control (AC) mode, the patient may increase the frequency (assist) in addition to the preset mechanical frequency (control). Each assist breath provides the preset mechanical tidal volume.

Indications for PEEP

Three major indications for PEEP are (1) intrapulmonary shunt and refractory hypoxemia, (2) decreased functional residual capacity (FRC) and lung compliance, and (3) auto-PEEP not responding to adjustments of ventilator settings.

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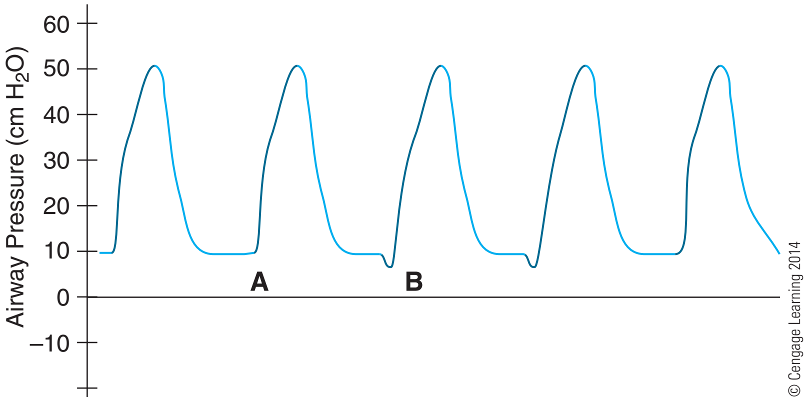

FIGURE 4-2 Positive end-expiratory pressure (PEEP). An assist/control pressure tracing with 10 cm H_2O of PEEP. (A) A controlled breath with PEEP. (B) An assisted breath with PEEP; note the negative deflection at the beginning of inspiration.

Intrapulmonary Shunt and Refractory Hypoxemia. The primary indication for PEEP is refractory hypoxemia induced by intrapulmonary shunting. This condition may be caused by a reduction of the FRC, atelectasis, or low V/Q mismatch (Tyler, 1983). Refractory hypoxemia is defined as hypoxemia that responds poorly to moderate to high levels of oxygen. A helpful *clinical* guideline for refractory hypoxemia is when the patient's PaO_2 is 60 mm Hg or lower at an F_IO_2 of 50% or higher. These values yield a calculated PaO_2/F_IO_2 (P/F) value of $\leq$120 mm Hg, which surpasses the threshold for ARDS ($<$ 200 mm Hg) (Wilkins et al., 2009).

Decreased FRC and Lung Compliance. A severely diminished FRC and reduced lung compliance greatly increase the alveolar opening pressure. If the patient is breathing spontaneously, a decreased lung compliance always increases the work of breathing and if severe enough can lead to fatigue of the respiratory muscles and ventilatory failure. Since PEEP increases the FRC, this pulmonary impairment may be prevented or improved by early application of PEEP.

Auto-PEEP. Air trapping may be caused by severe airflow obstruction or insufficient expiratory time. Bronchodilator therapy and pulmonary clearance are helpful to reduce airflow obstruction. Insufficient expiratory time may be corrected by increasing the peak flow, decreasing the frequency or tidal volume. Uncorrected air trapping may lead to auto-PEEP.

Auto-PEEP increases the work of breath triggering because the patient must overcome the auto-PEEP level, *plus* the sensitivity setting. For example, a patient has an auto-PEEP of 6 cm H_2O and the sensitivity is set at 2 cm H_2O below the end-expiratory baseline pressure. In this case, the patient would need to generate a total negative airway pressure of 8 cm H_2O (6 cm H_2O of auto-PEEP + 2 cm H_2O of sensitivity) to trigger a breath.

Auto-PEEP may be compensated by setting a PEEP level slight below the auto-PEEP level. This strategy raises the end-expiratory baseline pressure and reduces the breath-trigger effort. Refer to Figure 12-2 for an illustration.

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Physiology of PEEP

PEEP reinflates collapsed alveoli and supports and maintains alveolar inflation during exhalation. Once "recruitment" of these alveoli occurs and is sustained, PEEP decreases the threshold for alveolar opening and facilitates gas diffusion and oxygenation (Tyler, 1983).

Normally, the alveolar end-expiratory pressure equilibrates with atmospheric pressure (i.e., zero pressure) and the average pleural pressure is approximately –5 cm H_2O. Under these conditions, the alveolar distending pressure is 5 cm H_2O (alveolar-pleural). This distending pressure is sufficient to maintain a normal end-expiratory alveolar volume to overcome the elastic recoil of the alveolar wall. However, if the force of elastic recoil is increased due to a decrease in compliance, the alveolar volume will decrease. If the lung compliance continues to deteriorate, the elastic recoil forces can become great enough to completely overcome the normal alveolar distending pressure, resulting in alveolar collapse and intrapulmonary shunting. PEEP increases the alveolar end-expiratory pressure which decreases the pressure threshold for alveolar inflation. Re-expansion of the collapsed alveoli improves ventilation and reverses intrapulmonary shunting.

PEEP
↓
Decreases the pressure threshold for alveolar inflation
↓
Increases FRC
↓
Improves ventilation
↓
(1) Increases V/Q
(2) Improves oxygenation
(3) Decreases work of breathing

Complications of PEEP

Complications and hazards associated with PEEP include (1) decreased venous return and cardiac output, (2) barotrauma, (3) increased intracranial pressure, and (4) alterations of renal functions and water metabolism.

Decreased Venous Return. Assuming a normal intravascular volume, venous return to the right atrium is influenced by the difference in the central venous pressure and the negative pleural pressure that surrounds the heart. During PEEP, the pleural pressure becomes less negative and the pressure gradient between the central venous drainage and the right atrium will decrease resulting in a decreased venous return. This in turn results in a decreased cardiac output and hypotension (Qvist et al., 1975).

✓ Since PEEP increases both peak inspiratory pressures and mean airway pressures, it has the potential to decrease venous return and cardiac output.

Experience has shown that significant increases in the mean airway pressure are more likely to increase pleural pressures sufficiently to decrease venous return. Since PEEP increases both peak inspiratory pressures and mean airway pressures, it has the potential to decrease venous return and cardiac output. It is vital to closely monitor the patient receiving PEEP therapy for any drop in blood pressure, especially when PEEP is either first

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applied or increased to high levels. If PEEP decreases the blood pressure, first be sure that the patient is not hypovolemic (Shapiro et al., 1991). If the blood volume is adequate, then the PEEP should be decreased until an adequate blood pressure is reestablished.

✓ The detrimental effects of PEEP are dependent on the compliance characteristics of the patient.

A given amount of PEEP does not impede venous return to the same degree in different patients. If a patient has extremely low lung compliance, the airway pressure is less readily transmitted into the pleural space. In effect, the low lung compliance shields the pleural space from the full effects of the increased alveolar pressure. Patients with adult respiratory distress syndrome (ARDS) usually have a very low lung compliance and often require very high PEEP levels. However, despite high PEEP levels, hemodynamic instability is seldom a problem unless the patient has preexisting cardiovascular disease (Shapiro et al., 1991).

In contrast, if a patient has a normal or elevated lung compliance, the increased alveolar pressure due to the PEEP will more readily be transmitted into the pleural space. In other words, PEEP therapy in patients with normal or high lung compliance will more likely produce an elevated pleural pressure and therefore a decreased venous return (Shapiro et al., 1991).

✓ PEEP greater than 10 cm H_2O (or mean airway pressure $>$30 cm H_2O, peak inspiratory pressure $>$50 cm H_2O) is associated with an increased incidence of barotrauma.

Barotrauma. Barotrauma is lung injury that results from the hyperinflation of alveoli past the rupture point. Although each patient is different, a PEEP greater than 10 cm H_2O (or mean airway pressure $>$30 cm H_2O, or a peak inspiratory pressure $>$50 cm H_2O) is associated with an increased incidence of alveolar rupture or barotrauma (Bezzant et al., 1994; Slutsky, 1994). Alveolar rupture can produce pneumothorax, tension pneumothorax, pneumomediastinum, pneumopericardium, and pneumoperitoneum. Subcutaneous emphysema or crepitus of unknown cause should always be interpreted as a sign that barotrauma has occurred.

Since PEEP increases alveolar pressures and alveolar volumes, it has the potential to produce barotrauma (Petersen et al., 1983), especially when combined with volume-controlled ventilation. Therefore plateau pressures should be closely monitored and the therapist should be vigilant for signs of barotrauma.

✓ In patients with normal lung compliance, PEEP may increase the intracranial pressure due to impedance of venous return from the head.

Increased Intracranial Pressure. In patients with normal lung compliance, PEEP may raise the intracranial pressure (ICP) (normal 8 to 12 cm H_2O) due to an impedance of venous return from cerebral perfusion. However, in patients with ARDS or noncompliant lungs, transmission of the excessive pressure generated by PEEP is minimal and it does not cause as much adverse effect on a patient's ICP.

✓ Positive pressure ventilation can reduce the blood flow to the kidneys and affect their normal functions.

Alterations of Renal Functions and Water Metabolism. Kidneys play an important role in eliminating wastes, clearance of certain drugs, and regulating fluid, electrolyte, and acid-base balance. They are highly vascular and at any one time receive about 25% of the body's circulating blood volume (Brundage, 1992). Because of these characteristics, the kidneys are highly vulnerable to a decrease in blood flow, as would occur during positive pressure ventilation.

When perfusion to the glomeruli of the kidneys is decreased, filtration becomes less effective (Baer et al., 1992). Subsequently, the urine output is decreased, as the kidneys try to correct the perceived hypovolemic condition by retaining fluid. If hypoperfusion of the kidneys persists or worsens, renal failure may result.

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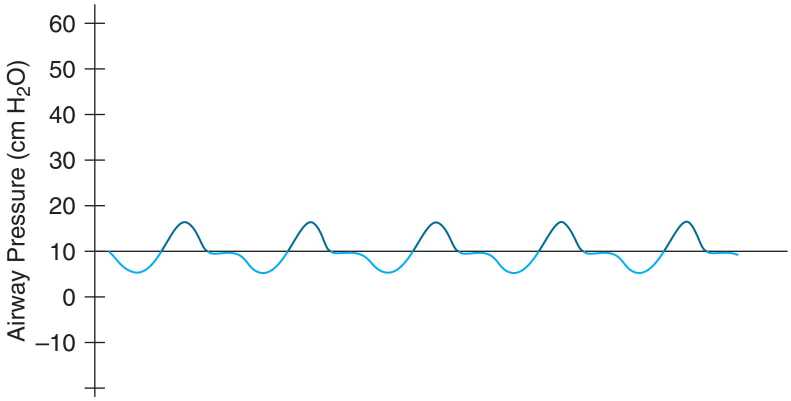

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FIGURE 4-3 Continuous positive airway pressure (CPAP) of 10 cm H_2O.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

eucapnic ventilation: The amount of ventilation needed to bring the patient's $PaCO_2$ to normal.

Continuous positive airway pressure (CPAP) is PEEP applied to the airway of a patient who is breathing spontaneously (Figure 4-3). The indications for CPAP are essentially the same as for PEEP with the additional requirement that the patient must have adequate lung functions that can sustain **eucapnic ventilation** documented by the $PaCO_2$. In adults, CPAP may be given via a face mask, nasal mask, or endotracheal tube. In neonates, nasal CPAP is the method of choice.

BILEVEL POSITIVE AIRWAY PRESSURE (BiPAP)

bilevel positive airway pressure (BiPAP): An airway pressure strategy that applies independent positive airway pressures (PAP) to both inspiration and expiration.

Bilevel positive airway pressure (BiPAP) allows the clinician to apply independent positive airway pressures to both inspiration and expiration. IPAP (inspiratory) and EPAP (expiratory) are used to define when the positive airway pressure is present. IPAP provides positive pressure breaths, and it improves ventilation and hypoxemia due to hypoventilation. EPAP is in essence CPAP, and it improves oxygenation by increasing the functional residual capacity and reducing intrapulmonary shunting.

Indications for BiPAP

BiPAP appears to be of value in preventing intubation of the end-stage COPD patient (Ambrosino et al., 1992; Confalonieri et al., 1994; Renston et al., 1994) and in supporting patients with chronic ventilatory failure (Strumpf, 1990). Other indications of BiPAP include patients with restrictive chest wall disease (Hill, 1992), neuromuscular disease (Ellis et al., 1987), and nocturnal hypoventilation (Carroll et al., 1988; Waldhorn, 1992). See Chapter 7, "Noninvasive Positive Pressure Ventilation."

✓ BiPAP appears to be of value in preventing intubation of the end-stage COPD patient and in supporting patients with chronic ventilatory failure.

Initial Settings

✓ The initial settings of IPAP and EPAP are 8 cm H_2O and 4 cm H_2O, respectively.

The BiPAP system may be used in one of three modes: spontaneous, spontaneous/timed, and timed. Mode selection depends on a patient's needs and ability to breath spontaneously. In general, if the patient is breathing spontaneously, the IPAP and EPAP may be initially set at 8 cm H_2O and 4 cm H_2O, respectively (ResMed, 1998a,b). The pressures are titrated based on needs, generally with a target of 5 to 7 mL/kg. Refer to Table 7-7 for titration of bilevel positive airway pressure. The spontaneous/timed mode is used as a backup mechanism and the frequency per min (f/min) is set two to five breaths below the patient's spontaneous frequency. In the timed mode, set IPAP and EPAP as above and the f/min slightly higher than the patient's spontaneous frequency. The % IPAP may be set at 33% or 50% for an I:E ratio of 1:2 or 1:1, respectively.

Adjustments of IPAP and EPAP

IPAP levels are generally determined by monitoring the patient's clinical and physiologic response to gradual changes of IPAP, rather than by directly measuring the volume delivered.

✓ IPAP may be increased in increments of 2 cm H_2O to enhance the "pressure boost" to improve alveolar ventilation, normalize $PaCO_2$, and reduce the work of breathing.

When the cardiopulmonary responses are positive, the IPAP may be increased in increments of 2 cm H_2O to enhance the "pressure boost" to improve ventilation, normalize $PaCO_2$, and reduce the work of breathing. Since IPAP does not provide volume-controlled ventilation, the volume delivered by IPAP is directly related to the IPAP and EPAP pressure gradient and the compliance characteristics of the lung/thorax system. The volume delivered is inversely related to the airflow resistance. In other words, a larger delivered volume may be obtained by (1) increasing the IPAP level, (2) decreasing the EPAP level, (3) increasing the compliance, and (4) reducing the airflow resistance.

✓ The EPAP should be increased by 2 cm H_2O increments to increase functional residual capacity and oxygenation in patients with intrapulmonary shunting.

The EPAP should be increased by 2 cm H_2O increments to increase functional residual capacity and oxygenation in patients with intrapulmonary shunting. When the EPAP is the same as the IPAP, CPAP results. It is not possible to increase the EPAP higher than the IPAP. Since IPAP and EPAP are methods to manipulate the airway pressures, all adverse effects of positive pressure ventilation and PEEP should be monitored. The patient should be advised to report any unusual chest discomfort, shortness of breath, or severe headache when using the BiPAP system.

✓ A BiPAP device can be used as a CPAP device by setting the IPAP and EPAP at the same level.

There are other similar modes that provide two CPAP or pressure levels (high pressure and low pressure), and the patient is allowed to breathe spontaneously without restriction. See airway pressure release ventilation (APRV) and biphasic positive positive airway pressure (biphasic PAP) in this chapter for details.

CONTROLLED MANDATORY VENTILATION (CMV)

control mode: In control mode, the ventilator delivers the preset tidal volume at a set time interval (time-triggered frequency).

With controlled mandatory ventilation (CMV), also known as continuous mandatory ventilation or **control mode,** the ventilator delivers the preset tidal volume at a time-triggered frequency (Figure 4-4). Since the ventilator controls both the patient's tidal volume and respiratory frequency, the ventilator "controls" the patient's minute

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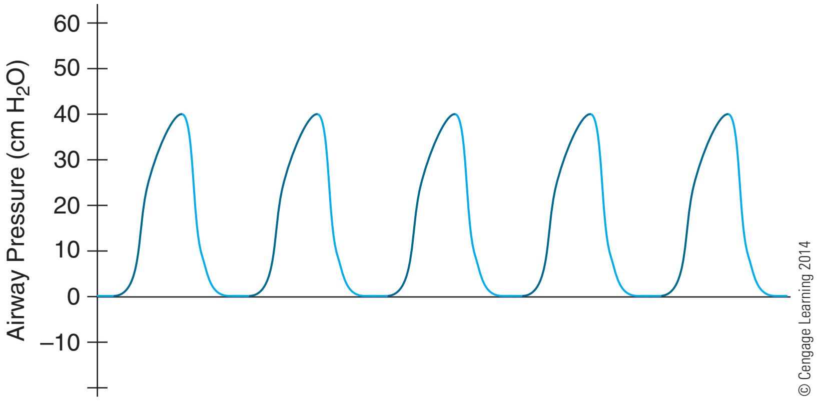

FIGURE 4-4 Control mode pressure tracing. The time intervals between mechanical breaths are equal when a control mode is used.

volume. In the control mode, a patient cannot change the ventilator frequency or breath spontaneously. For example, if the tidal volume and frequency of a ventilator are set at 800 mL and 10/min, respectively, the minute volume will be 8,000 mL.

✔ The RCP must recognize any spontaneous breathing efforts during control mode ventilation.

The control mode should only be used when the patient is properly medicated with a combination of sedatives, respiratory depressants, and neuromuscular blockers. The control mode ventilation should not be instituted by decreasing the ventilator's triggering sensitivity to the point that no amount of patient effort can trigger the ventilator into inspiration. The problem with this approach should be obvious since any spontaneous inspiratory effort would be like attempting to inspire through a completely obstructed airway. Regardless of how vigorous the patient's inspiratory effort is, no gas flow would be delivered to the patient until the ventilator automatically becomes time-triggered. If the control mode is improperly established in this way, it may not be physically harmful to the patient. However, it would most likely be psychologically devastating for the patient to realize that he or she has no control over his or her breathing requirements.

Indications for Control Mode

The control mode (with sedation and neuromuscular block) is sometimes indicated if the patient "fights" the ventilator in the initial stages of mechanical ventilatory support. "Fighting" or "bucking" the ventilator often means that the patient is severely distressed (e.g., hypoxia, pain) and vigorously struggling to breathe. Their rapid spontaneous inspiratory efforts become asynchronous with the ventilator's ability to provide an adequate inspiratory flow. The typical result is that the patient will be attempting to actively exhale while the ventilator is delivering a breath. This causes early termination of a mechanical breath due to high pressure limit cycling, which decreases the ventilator-delivered tidal volume.

Other indications for control mode ventilation include (1) tetanus or other seizure activities that interrupt the delivery of mechanical ventilation (Linton et al., 1992), (2) complete rest for the patient typically for a period of 24 hours (Perel et al., 1992), and (3) patients with a crushed chest injury in which spontaneous inspiratory efforts produce significant paradoxical chest wall movement (Burton et al., 1997).

TABLE 4-2 Characteristics of the Control Mode

Characteristic	Description
Type of breath	Each breath delivers a mechanical tidal volume.
Triggering mechanism	Every breath in the control mode is time-triggered.
Cycling mechanism	Inspiration is terminated by the delivery of a preset tidal volume (volume-cycled).

Complications of Control Mode

✓ In a sedated or apneic patient, the primary hazard of the control mode is the potential for apnea and hypoxia if the patient should become disconnected from the ventilator or the ventilator should fail to operate.

Since the patient's spontaneous respiratory drive will have been blunted with sedation and neuromuscular block in the control mode, the patient is totally dependent on the ventilator for ventilation and oxygenation. As a result, the primary hazard associated with the control mode is the potential for apnea and hypoxia if the patient should become accidentally disconnected from the ventilator or the ventilator should stop to operate due to mechanical or electrical failure.

Another physiologic problem with the control mode is the rapid disuse atrophy of diaphragm fibers. One study shows that a combination of 18 to 69 hours of complete diaphragmatic inactivity during mechanical ventilation results in marked atrophy of human diaphragm myofibers (Levine et al., 2008). Furthermore, prolonged mechanical ventilation leads to diaphragmatic oxidative injury, elevated proteolysis, and reduced function of the diaphragm (Betters et al., 2004). Because of the patient's dependence on the ventilator, the most important alarms in the control mode become those that alert the caregiver of any interruption in the patient's ventilation. The important ventilator alarms include the low exhaled volume alarm and the low inspiratory pressure alarm.

Table 4-2 summarizes the major characteristics of the control mode.

ASSIST/CONTROL (AC)

With the assist/control (AC) mode, the patient may increase the ventilator frequency (assist) in addition to the preset mechanical frequency (control). Each control breath provides the patient with a preset, ventilator-delivered tidal volume. Each assist breath also results in a preset, ventilator-delivered tidal volume. The assist control mode does not allow the patient to take spontaneous breaths (Figure 4-5).

Assist Control Triggering Mechanism

The mandatory mechanical breaths may be either patient-triggered by the patient's spontaneous inspiratory efforts (assist) or time-triggered by a preset frequency

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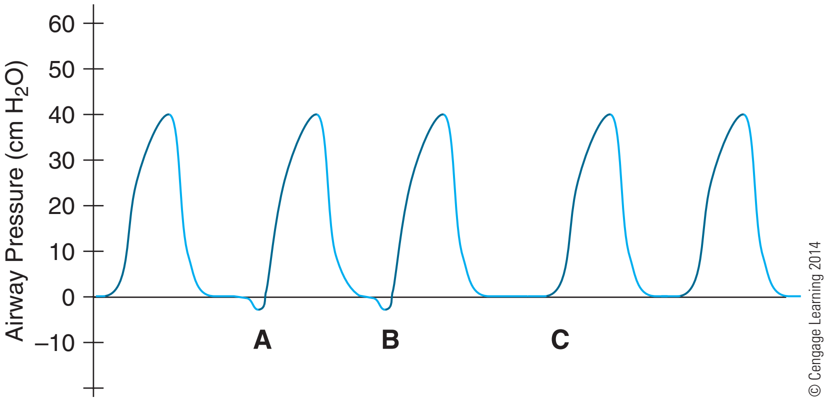

FIGURE 4-5 Assist/control mode pressure tracing. Each assisted or controlled breath triggers a mechanical tidal volume. (A) An assisted breath; note the negative deflection at the beginning of inspiration. (B) Another assisted breath that is initiated by the patient sooner than (A). (C) A controlled breath; note the absence of a negative deflection at the beginning of inspiration.

(control). If a breath is patient-triggered, it is referred to as an assisted breath; if a breath is time-triggered, the breath is referred to as a control breath.

For example, if the patient has a stable assist frequency of 12/min, then the patient is triggering breaths every 5 sec. If the control frequency is preset at 10/min, the ventilator would deliver time-triggered breaths every 6 sec. However, since the interval between the assisted breaths is shorter than 6 sec, no time-triggered breaths will be delivered. If however, the patient's spontaneous breaths were to decrease less than the preset control frequency, then the ventilator would begin delivering time-triggered breaths.

Assist Control Cycling Mechanism

Inspiration in the AC mode is terminated by volume cycling. When the preset tidal volume is delivered, the ventilator is cycled to expiration.

Indications for AC Mode

The AC mode is most often used to provide full ventilatory support for patients when they are first placed on mechanical ventilation. Full ventilatory support is defined as any ventilator mode that provides all of the work of breathing.

The AC mode is typically used for patients who have a stable respiratory drive (a stable spontaneous frequency of at least 10 to 12/min) and can therefore trigger the ventilator into inspiration. Essentially, the time-triggering control frequency is generally considered as a safety net to provide adequate ventilation in the event that the patient stops triggering the ventilator at an acceptable frequency (Sassoon et al., 1990). The generally accepted minimum control frequency in the AC mode is 2 to 4/min less than the patient's assist frequency, or a minimum control frequency of from 8 to 10/min.

TABLE 4-3 Characteristics of the Assist/Control Mode

Characteristic	Description
Type of breath	Each breath, assist or control, delivers a preset mechanical tidal volume.
Triggering mechanism	Mechanical breaths may be either patient-triggered (assist) or time-triggered (control).
Cycling mechanism	Inspiration is terminated either by the delivery of a preset tidal volume (volume-cycled) or by the high pressure limit (pressure-cycled).

Advantages of AC Mode

There are two primary advantages with the AC mode. First, the patient's work of breathing requirement in the AC is very small when the triggering sensitivity (pressure or flow) is set appropriately and the ventilator supplies an inspiratory flow that meets or exceeds the patient's inspiratory flow demand. The second advantage of AC is that, if the patient has an appropriate ventilatory drive, this mode allows the patient to control the frequency and therefore the minute volume required to normalize the patient's $PaCO_2$ (Kirby et al., 1988).

✓ The AC mode allows the patient to control the frequency and therefore the minute volume required to normalize the patient's $PaCO_2$.

Complications of AC Mode

The potential hazard associated with AC is alveolar hyperventilation (respiratory alkalosis). In two separate studies, the pH was found to be higher and the $PaCO_2$ was lower in the AC mode than the results obtained in the **intermittent mandatory ventilation (IMV)** mode (Culpepper et al., 1985; Hopper et al., 1985). If the patient's respiratory center is either injured or diseased, the patient may have an inappropriately high respiratory drive leading to an excessive assist frequency despite a low $PaCO_2$. If the patient is assisting at a high frequency (i.e., >20 to 25/min) and the tidal volume is preset at 10 to 15 mL/kg, this will usually result in hypocapnia and respiratory alkalosis.

intermittent mandatory ventilation (IMV): IMV is a mode in which the ventilator delivers control (mandatory) breaths and allows the patient to breathe spontaneously to any tidal volume the patient is capable of between the mandatory breaths.

Mechanical deadspace may be used in this situation, but it is generally considered safer to switch the patient to another mode of ventilation (e.g., SIMV) that limits the patient's ability to generate excessive minute volumes.

Table 4-3 summarizes the major characteristics of the assist/control mode.

INTERMITTENT MANDATORY VENTILATION (IMV)

Intermittent mandatory ventilation (IMV) is a mode in which the ventilator delivers control (mandatory) breaths and allows the patient to breathe spontaneously at any tidal volume the patient is capable of in between the mandatory breaths (Figure 4-6).

Historically, IMV was a separate circuit adapted to ventilators that were designed to provide either assist/control or control mode ventilation. As such, it was the first widely used mode that allowed partial ventilatory support (i.e., a mode that allowed

✓ Since IMV breaths are delivered at a frequency independent of the patient's spontaneous frequency, breath stacking may occur.

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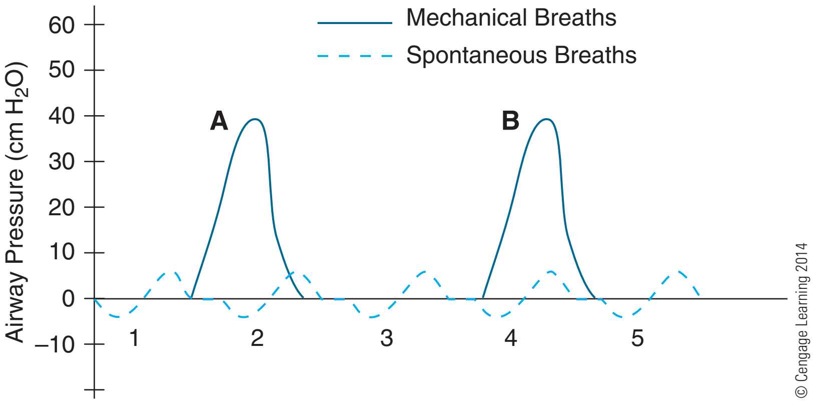

FIGURE 4-6 Intermittent mandatory ventilation (IMV) pressure tracing with two mandatory breaths and five anticipated spontaneous breaths (only three active). IMV mode may cause breath stacking since the mandatory breaths are delivered at a set time interval with no regard to the patient's breathing frequency. Mandatory breath (A) begins before the patient is ready for the anticipated spontaneous breath #2. Mandatory breath (B) begins shortly after the initiation of the anticipated spontaneous breath #4. The anticipated spontaneous breaths #2 and #4 did not occur as they turned into mechanical breaths during the mandatory cycle.

the patient to breathe spontaneously in addition to receiving ventilator-delivered breaths) (Heenan et al., 1980).

The primary complication associated with IMV was the random chance for breath stacking. This occurs when the patient is taking a spontaneous breath and the ventilator delivers a time-triggered mandatory breath at the same time. If this occurs, the patient's lung volume and airway pressure could increase significantly. Setting appropriate high pressure limits will reduce the risk of barotrauma in the event of breath stacking. As long as the breath stacking only occurs occasionally, the IMV mode is an acceptable mode of ventilation with few complications.

The sophistication of ventilator technology has progressed to the point that no new adult ventilators offer the IMV mode. Rather, all ventilators currently available have been designed to provide synchronized IMV (SIMV) (Shapiro et al., 1976).

SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION (SIMV)

synchronized intermittent mandatory ventilation (SIMV): SIMV is a mode in which the ventilator delivers control (mandatory) breaths to the patient at or near the time of a spontaneous breath. The mandatory breaths are synchronized with the patient's spontaneous breathing efforts so as to avoid breath stacking.

Synchronized intermittent mandatory ventilation (SIMV) is a mode in which the ventilator delivers either assisted breaths to the patient at the beginning of a spontaneous breath or time-triggered mandatory breaths. The mandatory breaths are synchronized with the patient's spontaneous breathing efforts so as to avoid breath stacking (Figure 4-7).

SIMV Mandatory Breath-Triggering Mechanism

The SIMV mandatory breaths may be either time-triggered or patient-triggered. The triggering mechanism is determined by whether or not the patient makes a

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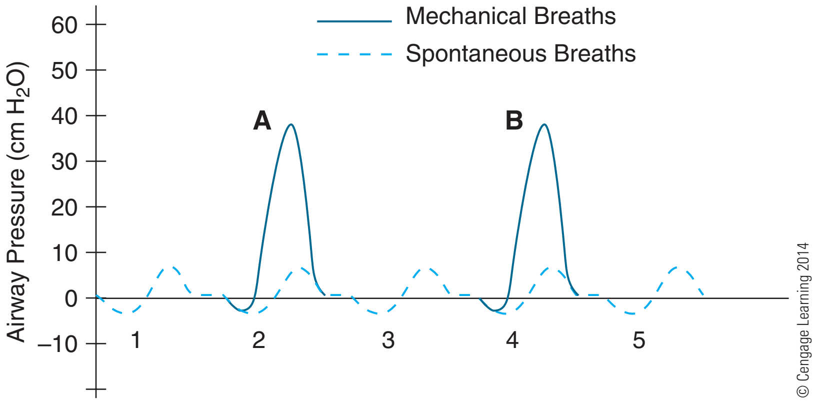

FIGURE 4-7 Synchronized intermittent mandatory ventilation (SIMV) pressure tracing with two mandatory breaths and five anticipated spontaneous breaths (only three active). SIMV mode does not cause breath stacking since the mandatory breaths are delivered slightly sooner or later than the preset time interval but within a time window. Mandatory breaths (A) and (B) occur during a spontaneous inspiratory effort. The anticipated spontaneous breaths #2 and #4 did not occur as they turned into mechanical breaths during the mandatory cycle.

spontaneous inspiratory effort just prior to the delivery of a time-triggered breath. For example, if the SIMV mandatory frequency is set at 10/min, then the ventilator would time-trigger a breath every 6 sec if the patient is not attempting to inspire spontaneously. However, if the patient is breathing spontaneously between the mandatory breaths, and if by random chance, the patient begins to inspire just prior to the point at which the ventilator would be expected to time-trigger, then the ventilator senses this spontaneous effort and delivers the mandatory breath as an assisted patient-triggered breath. The mandatory breath, whether time- or patient-triggered, is controlled by all applicable mechanical tidal volume settings.

Synchronization Window. The time interval just prior to time triggering in which the ventilator is responsive to the patient's spontaneous inspiratory effort is commonly referred to as the *synchronization window* (Sassoon et al., 1991). Although the exact time interval of the synchronization window is slightly different from manufacturer to manufacturer, 0.5 sec is representative. For example, given an SIMV mandatory frequency of 10/min, the ventilator would be expected to time trigger every 6 sec. If the synchronization window is 0.5 sec, then at 5.5 sec from the beginning of the previous mandatory breath, the ventilator automatically becomes sensitive to any spontaneous inspiratory effort, i.e., the synchronization window becomes active. If the patient makes a spontaneous inspiratory effort when the synchronization window is active, the ventilator is patient-triggered to deliver an assisted mandatory breath. Patient triggering may be based either on pressure or flow. If, however, no spontaneous inspiratory effort exists while the synchronization window is active, the ventilator will time trigger when the full time-triggering interval elapses.

SIMV Spontaneous Breath-Triggering Mechanism

In between the mandatory breaths, SIMV permits the patient to breathe spontaneously to any tidal volume the patient desires. The gas source for spontaneous

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✓ Spontaneous frequency and tidal volume taken by the patient in the SIMV mode are totally dependent on the patient's breathing effort.

breathing in the SIMV mode is typically supplied by a demand valve. The demand valve is always patient-triggered, either by pressure or flow depending on the ventilator.

It is important to understand that the spontaneous breaths taken by the patient in the SIMV mode are truly spontaneous. The ventilator provides the humidified gas at the selected F_IO_2, but the spontaneous frequency and spontaneous tidal volume are totally dependent on the patient's breathing effort.

Indications for SIMV Mode

✓ The primary indication for SIMV is to provide partial ventilatory support to the patient.

The primary indication for SIMV is to provide partial ventilatory support, i.e., a desire to have the patient actively involved in providing part of the minute volume. In a practical sense, when a patient is first placed on ventilatory support, full ventilatory support is appropriate to provide a period of rest, typically for the first 24 hours. After this initial period of full ventilatory support, it is a typical practice to place the patient on a trial run of partial ventilatory support with SIMV. It is customary to ease the patient from full support to partial support by gradually decreasing the mandatory frequency as tolerated by the patient. This depends on, of course, reversal of the clinical conditions that committed the patient to the ventilator in the first place.

Advantages of SIMV Mode

Since SIMV promotes spontaneous breathing and use of respiratory muscles, SIMV (1) maintains respiratory muscle strength/avoids muscle atrophy, (2) reduces ventilation to perfusion mismatch, (3) decreases mean airway pressure, and (4) facilitates weaning.

Maintains Respiratory Muscle Strength/Avoids Muscle Atrophy. SIMV helps to maintain respiratory muscle activity and strength. Patients maintained in full ventilatory support for extended periods tend to experience partial loss of ventilatory muscle strength. This can be minimized by using the respiratory muscles during spontaneous breathing (Zelt et al., 1972).

Reduces Ventilation to Perfusion Mismatch. Deadspace ventilation (i.e., high ventilation to low perfusion) is typical in the upper zone of the lungs because pulmonary perfusion is gravity-dependent and favors the lower lung zone. This problem is intensified during positive pressure ventilation, as the lung units in the upper zone are hyperinflated. Spontaneous breathing during SIMV tends to distribute the spontaneous tidal volume more evenly thus reducing alveolar deadspace ventilation (Weisman et al., 1983).

Decreases Mean Airway Pressure. The mean airway pressure is directly related to the peak inspiratory pressure and inspiratory time (Note: Two other factors are frequency and positive end-expiratory pressure). Since spontaneous breaths during SIMV have a lower peak inspiratory pressure and inspiratory time, SIMV tends to have a lower mean airway pressure.

TABLE 4-4 Characteristics of the Synchronized IMV Mode

Characteristic	Description
Type of breath	The ventilator delivers mechanical tidal volume at a preset frequency. The patient may breathe spontaneously between mandatory breaths.
Triggering mechanism	Mandatory breaths may be either time-triggered or patient-triggered. Spontaneous breaths are patient-triggered (i.e., the demand flow valve opens in response to the patient's spontaneous inspiratory effort).
Cycling mechanism	The mandatory breaths are volume-cycled. The patient controls spontaneous frequency and volume.

The mean airway pressure is an important consideration because the greater the mean airway pressure, the greater the potential for a reduced venous return, cardiac output, and arterial perfusion pressure. Reduction of the mean airway pressure during SIMV indirectly enhances the patient's cardiovascular functions (Wilkins et al., 2003).

Facilitates Weaning. SIMV facilitates weaning due to its ability to decrease the mandatory frequency in small increments. This may offer some advantage to those "hard-to-wean" patients who cannot tolerate an abrupt decrease of the mechanical frequency or spontaneous breathing trial (Downs et al., 1973).

Complications of SIMV Mode

Prior to PSV (pressure support ventilation), the approach of SIMV weaning is to provide a spontaneous breathing workload that gradually increases a patient's muscle strength and endurance. The primary disadvantage associated with SIMV is the desire to wean the patient too rapidly, leading first to a high work of spontaneous breathing and ultimately to muscle fatigue and weaning failure. Without PSV, the best practice is to decrease the SIMV mandatory frequency slowly and monitor the patient closely for signs of fatigue (Wilkins et al., 2003).

Table 4-4 summarizes the major characteristics of the synchronized IMV mode.

MANDATORY MINUTE VENTILATION (MMV)

mandatory minute ventilation (MMV): MMV is a feature of some ventilators that causes an increase of the mandatory frequency (Note: In Hamilton Veolar, the pressure support level), when the patient's spontaneous breathing level becomes inadequate. This compensation by the ventilator ensures a safe minimal minute ventilation.

Mandatory minute ventilation (MMV), also called minimum minute ventilation, is a feature of some ventilators that provides a predetermined minute ventilation when the patient's spontaneous breathing effort becomes inadequate. For example, an apnea episode (lack of spontaneous breathing) may cause the actual minute ventilation to drop below the preset level. When this occurs, the mandatory frequency is increased automatically to compensate for the decrease in minute ventilation caused by the apnea. This compensation by the ventilator ensures a desired minute ventilation.

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MMV is an additional function of the SIMV mode and is intended to prevent hypercapnia by "automatically" ensuring that the patient receives a minimum preset minute volume. It is especially useful in preventing hypoventilation and respiratory acidosis in the final stages of weaning with SIMV when the patient's spontaneous breathing is assuming a significant portion of the total minute volume.

For example, a patient may have been weaned down to a mandatory SIMV frequency of 4/min with a mandatory tidal volume of 800 mL; the patient's ventilator-delivered minute volume would then be 3.2 L/min ($\dot{V}_E = f \times V_T$). If this patient's spontaneous minute volume is 6 L/min, then the total minute volume is the sum of the ventilator-delivered minute volume and the spontaneous breathing minute volume (9.2 L/min in this example). If the patient's spontaneous minute volume suddenly decreases by a significant amount, or if the patient becomes apneic, then without MMV the reduced minute volume would cause hypercapnia and respiratory acidosis. However, on MMV-equipped ventilators, a decrease in the patient's spontaneous minute volume would trigger an automatic increase in the ventilator's mandatory frequency.

The way that MMV functions on the majority of ventilators is that a desired minimum minute volume is preset on the ventilator—usually only slightly less than the minute volume required to "normalize" the $PaCO_2$. The ventilator then measures the total minute volume and compares it with the preset minimum minute volume. As long as the patient's total minute volume equals or exceeds the preset minimum minute volume, the MMV function is not activated. However, if the patient's spontaneous minute volume decreases to the point that the total minute volume becomes less than the preset mandatory minute volume, then the ventilator will automatically increase the SIMV mandatory frequency until it reaches the preset mandatory minute volume.

In the MMV mode, it is important to monitor not only the patient's spontaneous minute volume, but also the patient's estimated spontaneous alveolar minute volume. The reason for this is that if the patient becomes distressed, the tendency is to increase the spontaneous frequency at the expense of a decreased tidal volume (i.e., the patient will typically adopt the spontaneous breathing pattern that minimizes the work of breathing). A minute volume supported by a rapid frequency and low tidal volume may avert the MMV function but at the same time provides a significant amount of deadspace ventilation. This results in a decreased alveolar minute volume.

✓ A minute volume supported by rapid frequency and low tidal volume (e.g., distressed patient) may avert the MMV function but at the same time provides a significant amount of deadspace ventilation. This results in a decreased alveolar minute volume.

Perhaps the most efficient method of ensuring that this condition does not occur is to set the high frequency alarm at approximately 10/min greater than the patient's "baseline" spontaneous frequency.

Although MMV operates in the manner previously described on most ventilators, one exception is seen in the Hamilton Veolar ventilator. Selecting the MMV mode on this ventilator automatically places the patient in a "pure" pressure support mode (i.e., every breath is a spontaneous pressure-supported breath and no mandatory breaths are given). A minimum desired mandatory minute volume is selected and the ventilator automatically compares the patient's total minute volume with the preset minimum minute volume. On the Veolar, if the patient's total minute

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TABLE 4-5 Characteristics of the Mandatory Minute Ventilation Mode

Characteristic	Description
Type of breath	The ventilator increases the mandatory frequency (Note: Hamilton Veolar increases the pressure support level).
Triggering mechanism	Increase of the mandatory frequency (or the pressure support level in the Hamilton Veolar) is triggered when the actual minute volume is less than the preset minimal minute volume.
Cycling mechanism	All mandatory breaths are volume-cycled. Patients control their own spontaneous frequency and volume.

volume is less than the preset minimum minute volume, the ventilator automatically increases the pressure support level until the minimum minute volume is obtained (Wilkins et al., 2003).

Table 4-5 summarizes the major characteristics of the mandatory minute ventilation mode.

PRESSURE SUPPORT VENTILATION (PSV)

PSV lowers the work of spontaneous breathing and augments the spontaneous tidal volume.

Pressure support ventilation (PSV) is used to lower the work of spontaneous breathing and augment a patient's spontaneous tidal volume. When PSV is used with SIMV, it significantly lowers the oxygen consumption requirement presumably due to the reduced work of breathing (Kanak et al., 1985).

PSV applies a preset pressure plateau to the patient's airway for the duration of a spontaneous breath (Figure 4-8). Pressure-supported breaths are considered spontaneous because (1) they are patient-triggered, (2) the tidal volume varies with the

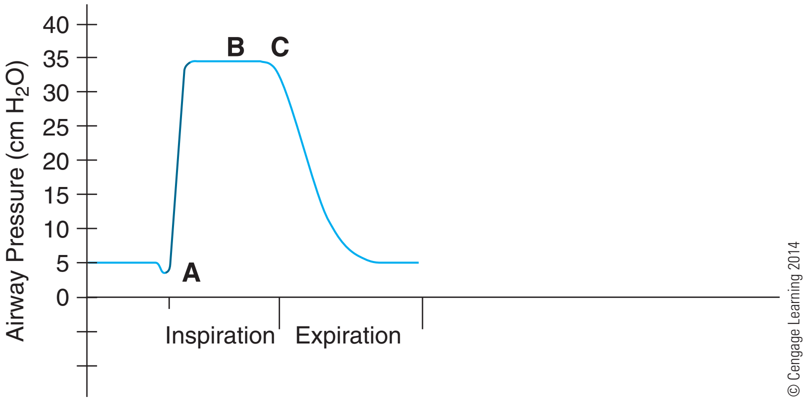

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FIGURE 4-8 Pressure support ventilation (PSV) with PEEP of 5 cm H_2O. (A) Inspiratory effort; (B) Pressure support plateau of 30 cm H_2O (peak inspiratory pressure of 35 cm H_2O, PEEP of 5 cm H_2O); (C) Beginning expiratory phase when the inspiratory flow drops to 25% (or other predetermined %) of its peak flow.

patient's inspiratory flow demand, (3) inspiration lasts only for as long as the patient actively inspires, and (4) inspiration is terminated when the patient's inspiratory flow demand decreases to a preset minimal value. PSV can be used in conjunction with spontaneous breathing in any ventilator mode.

A pressure-supported breath is therefore patient-triggered, pressure-limited, and flow-cycled. It is pressure-limited because the maximum airway pressure cannot exceed the preset pressure support level. It is flow-cycled because a pressure-supported breath cycles to expiration when the flow reaches a minimal level.

It is important to understand how the pressure plateau is created and maintained. Essentially, when the pressure-supported breath is patient-triggered (either by pressure or flow), the ventilator demand valve generates a flow high enough to rapidly increase the airway pressure to the preset pressure limit and then maintain the pressure plateau (via servo control and demand valve) for the duration of the patient's spontaneous inspiratory effort. Typically, the flow pattern associated with pressure support is a steeply descending tapered flow pattern. As previously described, the demand valve flow terminates when it decreases to a preset lower flow limit. The point at which flow cycling occurs varies with different ventilators but 5 L/min or 25% of peak flow are two examples.

Indications for PSV Mode

Pressure support is commonly applied in the SIMV mode when the patient takes spontaneous breaths. Pressure support is not active during the mandatory breaths. Pressure support has been advocated as a stand-alone mode by some clinicians; however, this requires close monitoring because as a stand-alone mode, every breath is patient-triggered.

✓ Pressure support (1) ↑ spontaneous tidal volume, (2) ↓ spontaneous frequency, and (3) ↓ work of breathing.

Pressure support is typically used in the SIMV mode to facilitate weaning in a difficult-to-wean patient. In this application, pressure support (1) increases the patient's spontaneous tidal volume, (2) decreases the patient's spontaneous frequency, and (3) decreases the work of breathing.

✓ The level of pressure support is titrated until (1) spontaneous tidal volume = 10 to 15 mL/kg or (2) spontaneous frequency < 25/min.

These three effects have been used to titrate the proper level of pressure support. For example, one physician may increase the pressure support level until a desired spontaneous tidal volume is achieved (e.g., 10 to 15 mL/kg). Another physician may increase the pressure support level until the patient's spontaneous frequency decreases to a target value (usually 25/min or less) (MacIntyre, 1987).

The third endpoint for the pressure support level is to decrease the work of breathing (MacIntyre, 1986). This approach is probably less commonly used for the patient in immediate respiratory distress, but is more often used as a "routine" method to decrease the work of breathing. Since an endotracheal tube increases the airway resistance and the work of breathing, pressure support has been used successfully to overcome this gas flow resistance. The airway resistance on most modern ventilators may be obtained easily, but in ventilators not equipped with this function, the following equation may be used to estimate the airway resistance:

✓ See Appendix 1 for example.

$$\text{Airway Resistance} = \frac{(\text{Peak Inspiratory Pressure} - \text{Plateau Pressure})}{\text{Mean Flow}}$$

Table 4-6 summarizes the major characteristics of the pressure support ventilation mode.

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TABLE 4-6 Characteristics of the Pressure Support Ventilation Mode

Characteristic	Description
Type of breath	Pressure-supported breaths are considered spontaneous. (Note: Pressure support may be applied in any mode that permits spontaneous breathing such as SIMV.)
Triggering mechanism	Pressure-supported breaths are patient-triggered.
Cycling mechanism	Pressure-supported breaths are technically flow-cycled by a minimum spontaneous inspiratory flow threshold. This minimum inspiratory flow is controlled entirely by the patient's spontaneous inspiratory flow demand.

Notes: The tidal volume delivered by a pressure-supported breath is influenced both by the pressure support level (cm H_2O) and the patient's spontaneous inspiratory flow demand. The inspiratory time of the pressure-supported breath is also completely controlled by the patient's spontaneous inspiratory flow demand.

ADAPTIVE SUPPORT VENTILATION (ASV)

adaptive support ventilation (ASV): A mode of ventilation that changes the number of mandatory breaths and pressure support level according to the patient's breathing pattern.

The **adaptive support ventilation (ASV)** is a dual control mode that provides a mandatory minute ventilation. The ventilator measures the dynamic compliance and expiratory time constant to adjust the mechanical tidal volume and frequency for a target minute ventilation. The optimal tidal volume is calculated by dividing the minute ventilation by the optimal frequency in terms of the lowest work of breathing. Basically, ASV uses the Otis Equation to calculate the optimal frequency that corresponds with the lowest work of breathing (Chen et al., 2008; Otis et al., 1950). Figure 4-9 shows the selection of optimal frequency based on the lowest total work of breathing (WOB tot) (Lotti et al., 2002).

With ASV mode, the therapist inputs the patient's body weight and the desired percent minute volume. The body weight is used to estimate the deadspace volume and to calculate the alveolar volume. For an estimated minute ventilation requirement for a patient, the ventilator uses predetermined settings of 100 mL/min/kg for adults and 200 mL/min/kg for children. The therapist may select the percent minute volume, ranging from 20% to 200% of the predetermined adult or child setting. For example, if 160% is selected for an adult, the minute ventilation delivered by the ventilator will be about 160 mL/min/kg.

Once the target minute ventilation is set, the ventilator uses test breaths to measure the system compliance, airway resistance, and any intrinsic PEEP. Following determination of these variables, the ventilator selects and provides the frequency, inspiratory time, I:E ratio, and high pressure limit for mandatory and spontaneous breaths.

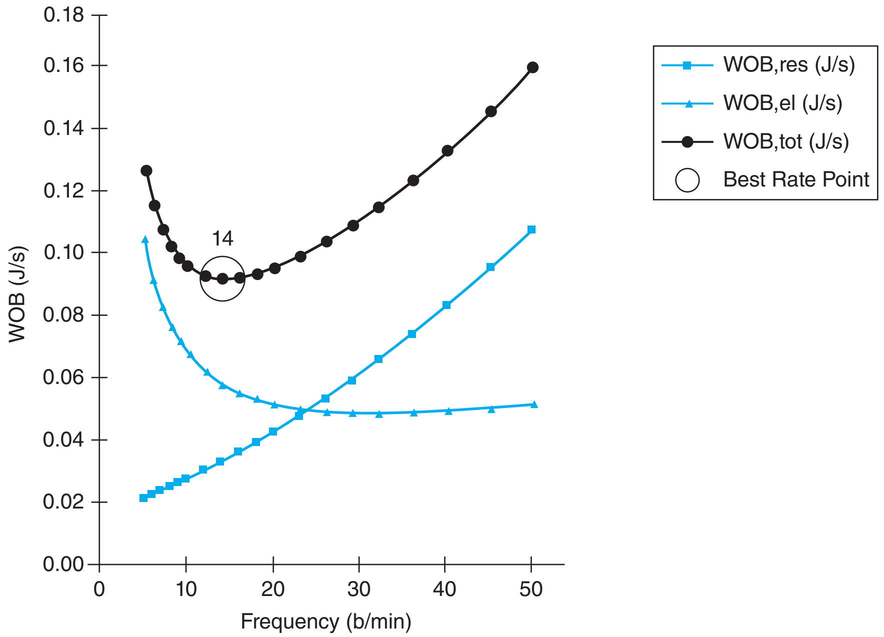

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FIGURE 4-9 Work of breathing model based on the Otis Equation. Note the respiratory rate of 14 corresponds with the lowest point on the total work of breathing (WOB, tot) curve. (WOB = work of breathing, res = resistive, el = elastic)

If there is no spontaneous triggering effort, the ventilator determines and provides the mandatory frequency, tidal volume, and high pressure limit needed to deliver the preselected tidal volume, inspiratory time, and I:E ratio. As the patient begins to trigger the ventilator, the number of mandatory breaths decreases and the pressure support level increases until a calculated tidal volume is able to provide adequate alveolar volume (i.e., tidal volume = alveolar volume + 2.2 mL/kg of deadspace volume).

PROPORTIONAL ASSIST VENTILATION (PAV)

Proportional assist ventilation (PAV): A mode that uses variable pressure to provide pressure support. The variable pressure is in proportion to the patient's pulmonary characteristics (elastance and airflow resistance) and demand (volume or flow).

Proportional assist ventilation (PAV) (Evita 4, Dräger Medical) and proportional pressure support are similar modes of ventilation. With PAV, there is no target flow, volume, or pressure during mechanical ventilation. The pressure used to provide the pressure support is variable and is in proportion to the patient's pulmonary characteristics (elastance and airflow resistance) and demand (volume or flow) (Appendini et al., 1999). PAV is set to overcome 80% of the elastance and airflow resistance. For example, at 40 cm H_2O/L of elastance, 32 cm H_2O of pressure is generated to provide 1 L of volume.

✓ PAV may be flow assist or volume assist and it is active in assist breaths only.

PAV may be flow assist (FA) or volume assist (VA). In FA, the applied pressure is provided to meet the patient's inspiratory flow demand. FA reduces the inspiratory effort needed to overcome airflow resistance (Appendini et al., 1999). VA occurs when PAV provides the pressure to meet the patient's volume requirement. VA

TABLE 4-7 Characteristics of the Proportional Assist Ventilation Mode

Characteristic	Description
Type of breath	PAV occurs during assisted breaths only
Triggering mechanism	Pressure- or flow-triggered
Cycling mechanism	PAV terminates once the patient's volume or flow demand is met

reduces the inspiratory effort needed to overcome systemic elastance such as restrictive lung defects (Navalesi et al., 1996).

PAV is achieved by a positive feedback control that amplifies airway pressure in proportion to instantaneous inspiratory flow and volume. Unlike the traditional modes of mechanical ventilation, PAV changes with the patient's breathing effort. The advantage of PAV is its ability to track changes in breathing effort over time. By varying the pressure to augment flow and volume, a more uniform breathing pattern becomes possible (Bigatello et al., 1997). PAV has been reported to provide mechanical ventilation while promoting patient-ventilator synchrony (Younes, 1992). However, in conditions where the elastance or airflow resistance shows sudden improvement, the pressure provided by PAV may be too high. This may lead to overdistension, increased air trapping, and barotrauma.

✓ In conditions where the elastance or airflow resistance shows sudden improvement, the pressure provided by PAV may be too high. This may lead to overdistension, increased air trapping, and barotrauma.

In terms of physiologic response, PAV improves ventilation and reduces the neuromuscular drive and work of breathing in ventilator-dependent patients with COPD. When PAV is used with CPAP, the reduction of inspiratory muscle work reaches values close to those found in normal subjects. Exercise tolerance may be improved with this strategy of combining PAV with CPAP (Appendini et al., 1999; Dolmage & Goldstein, 1997). Table 4-7 summarizes the major characteristics of the PAV mode.

VOLUME-ASSURED PRESSURE SUPPORT (VAPS)

volume-assured pressure support (VAPS): A mode of ventilation that assures a stable tidal volume by incorporating inspiratory pressure support ventilation (PSV) with conventional volume-assisted cycles (VAV).

Volume-assured pressure support (VAPS) is available in the Bird 8400 STi and TBird ventilators. It is similar to pressure augmentation in the Bear 1000 ventilator and is also known as volume-assisted pressure support.

VAPS incorporates inspiratory pressure support ventilation (PSV) with conventional volume-assisted cycles (VAV). This combination provides an optimal inspiratory flow during assisted/controlled cycles, reducing the patient's work of breathing commonly seen during VAV. Unlike typical PSV, VAPS assures stable tidal volume in patients with irregular breathing patterns (Amato et al., 1992).

In VAPS, the therapist must preset the desired minimum tidal volume and the pressure support level. During VAPS, the mechanical breaths may be patient- or

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time-triggered. Once a breath is triggered, the ventilator tries to reach the pressure support level as soon as possible. The delivered volume is then compared with the preset tidal volume for further action by the ventilator.

If the delivered volume equals the preset volume, the breath is considered a pressure support breath. Since pressure support breaths are dependent on the patient effort, the delivered volume may be larger than the preset volume. It is essential to set the pressure support level that provides a volume that is lower than the preset volume. On the other hand, if the delivered volume falls short of the preset volume, the ventilator switches from a pressure-limited breath to a volume-limited breath. This results in a longer inspiratory time (at a constant flow) until the preset volume is delivered.

✓ VAPS may prolong the inspiratory time. Patients with airflow obstruction should be monitored closely in order to prevent air trapping and other related side effects.

Since VAPS may prolong the inspiratory time automatically, patients with airflow obstruction should be monitored closely in order to prevent air trapping and other undesirable cardiovascular effects associated with prolonged inspiratory time.

PRESSURE-REGULATED VOLUME CONTROL (PRVC)

pressure-regulated volume control (PRVC): A mode of ventilation that provides volume-controlled breaths with the lowest pressure possible by altering the flow and inspiratory time.

Pressure-regulated volume control (PRVC) is first available in the Siemens 300. Similar modes to PRVC in subsequent ventilators are known as: adaptive pressure control (Servo-I, Maquet), AutoFlow (Evita XL, Drager), adaptive pressure ventilation (Galileo, Hamilton), volume control + (Puritan Bennett, Tyco), volume targeted pressure control and pressure controlled volume guaranteed (Engstrom, General Electric) (Mireles-Cabodevila et al., 2009). In the Siemens 300 ventilator, PRVC is only active during CMV whereas in other ventilators, this type of dual control mode is also active in SIMV. Table 4-8 outlines the characteristics of the PRVC mode.

PRVC is used primarily to achieve volume support while keeping the peak inspiratory pressure (PIP) at a lowest level possible. This is achieved by altering the peak flow and inspiratory time in response to changing airway or compliance characteristics.

✓ PRVC provides volume support while keeping the PIP at a lowest level possible by altering the peak flow and inspiratory time in response to changing airway or compliance characteristics.

For example, Equation (1) shows that at constant flow, the PIP is increased due to increasing airflow resistance. Note that the increasing airflow resistance may be due

TABLE 4-8 Characteristics of the Pressure-Regulated Volume Control Mode

Characteristic	Description
Type of breath	CMV in Siemens 300; SIMV in other ventilators
Triggering mechanism	Time-triggered or patient-triggered
Cycling mechanism	Volume-cycled under normal operating conditions

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to increasing airway resistance (nonelastic resistance) or decreasing lung compliance (elastic resistance). Since PIP and flow have a direct mathematical relationship, PRVC lowers the flow to reduce the driving pressure. See Equation (2).

$$(1)\ \uparrow \text{Airflow Resistance (nonelastic or elastic)} = \uparrow \text{PIP / Flow}$$

$$(2)\ \uparrow \text{Airflow Resistance (nonelastic or elastic)} = \text{PIP} / \downarrow \text{Flow}$$

To compensate for a lower inspiratory flow, PRVC prolongs the inspiratory time to deliver the target volume ($V_T = \uparrow \text{Constant Flow} \times \downarrow \text{I Time}$).

Automode

automode: This mode provides time-triggered, PRVC breaths when prolonged apnea is detected (12, 8, and 5 sec in adult, pediatric, and neonatal modes respectively).

Automode is a feature of the Siemens 300A ventilator and it combines PRVC and volume support. This mode alters between time-cycled and flow-cycled breaths depending on the degree of patient effort. If there is no spontaneous triggering effort for a time period (i.e., apnea for 12, 8, and 5 sec in adult, pediatric, and neonatal modes, respectively), the ventilator provides PRVC and the breaths are time-triggered. The delivered volume is preset with a variable PIP up to the high pressure limit.

When the patient has two consecutive breaths that trigger the mechanical breaths, the automode switches to volume support in which all breaths become patient-triggered, pressure-limited, and flow-cycled.

ADAPTIVE PRESSURE CONTROL (APC)

adaptive pressure control (APC): A mode of pressure-controlled breath that utilizes closed-loop control of the pressure setting to maintain a minimum delivered tidal volume.

Adaptive pressure control (APC) offers a dual-control mechanism that combines the functions of volume ventilation (stable tidal volume) with the functions of pressure ventilation (via variable flow). Adaptive pressure control is a pressure-controlled breath that uses variable inflation pressures (closed-loop control of the pressure) to deliver a minimum targeted tidal volume. APC does not guarantee a constant tidal volume. Since the inflation pressure is variable, as the patient's inspiratory effort increases the inflation pressure is reduced. This is a concern because the ventilator cannot distinguish between improved pulmonary compliance and increased patient effort (Branson et al., 2007, Mireles-Cabodevila et al., 2009). Increasing patient's breathing effort due to hypoxia or pain may potentially create a greater work of breathing (due to decreasing inflation pressure).

VOLUME VENTILATION PLUS (VV+)

volume ventilation plus (VV+): An option that combines volume control plus and volume support.

Volume ventilation plus (VV+) is available in the Puritan Bennett 840® ventilator (Tyco Healthcare). It is an option that combines two different dual mode volume-targeted breath types: volume control plus and volume support.

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Volume Control Plus (VC+)

✓ Following a test breath, the target pressures for subsequent breaths are adjusted accordingly to compensate for any tidal volume differences (Set V_T − Delivered V_T).

✓ In VC+, active spontaneous breaths are allowed during the inspiratory phase of a mandatory breath.

Volume control plus (VC+) is used to deliver mandatory breaths during AC and SIMV modes of ventilation. VC+ is intended to provide a higher level of synchrony than standard volume control ventilation.

In VC+, the clinician sets the target tidal volume and inspiratory time. The ventilator delivers a single test breath using standard volume and decelerating flow and plateau to determine the relative compliance. The target pressures for subsequent breaths are adjusted accordingly to compensate for any tidal volume differences (Set V_T − Delivered V_T). Flow is adjusted automatically to reduce the likelihood of inadequate flow or aggressive flow demand.

Active spontaneous breaths are allowed during the inspiratory phase of a mandatory breath by way of a pressure control style of breath and the use of an active exhalation valve. Excessive pressure caused by breathing or coughing is vented, thus maintaining synchrony.

Volume Support (VS)

✓ In VS, the clinician sets the target tidal volume and the ventilator uses variable pressure support levels to provide the target tidal volume.

Volume support (VS) is intended to provide a control tidal volume and increased patient comfort. Weaning from anesthesia is a common application for VS.

In VS, the clinician sets the target tidal volume but not the inspiratory time or mandatory frequency. The ventilator delivers a single spontaneous pressure support type of breath and uses variable pressure support levels to provide the target tidal volume. During weaning or awakening from anesthesia, the patient assumes a higher spontaneous tidal volume and the ventilator decreases the pressure support level accordingly. When the spontaneously tidal volume decreases, the ventilator increases the pressure support level automatically to maintain the target tidal volume.

During VS, the ventilator frequency and minute ventilation are determined by the triggering effort of the patient. The inspiratory time is determined by the patient respiratory demand.

PRESSURE-CONTROLLED VENTILATION (PCV)

pressure-controlled ventilation (PCV): A pressure plateau is created at the beginning of inspiration and the pressure is maintained for a preset inspiratory time. PCV can minimize the airway pressures while providing support to oxygenation and ventilation.

In **pressure-controlled ventilation (PCV),** the pressure-controlled breaths are time-triggered by a preset frequency. Once inspiration begins, a pressure plateau is created and maintained for a preset inspiratory time. Pressure-controlled breaths are therefore time-triggered, pressure-limited, and time-cycled.

PCV has some functional similarities to pressure support ventilation, but they have very different indications. Pressure-controlled breaths are time-triggered by a preset frequency, and, as in the control mode, the patient should be sedated. Once a pressure-controlled breath has been time-triggered, a pressure plateau is created and maintained by servo-controlled inspiratory flow in a manner similar to pressure support. Recall that the pressure plateau in pressure support is

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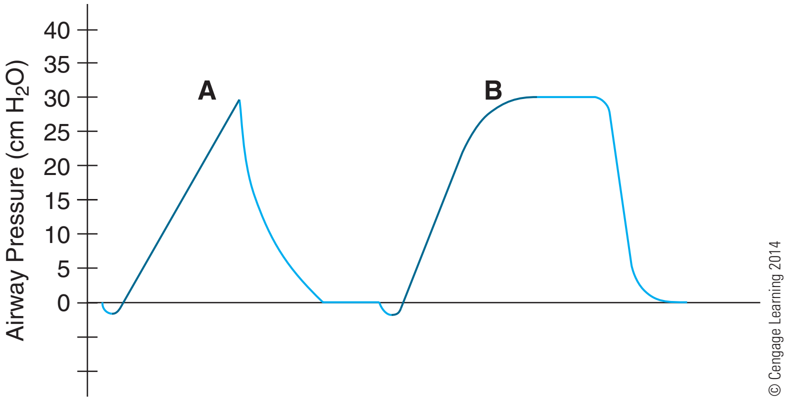

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FIGURE 4-10 (A) Pressure tracing of a volume-controlled mechanical breath; (B) Pressure tracing of a pressure-controlled mechanical breath; note the prolonged inspiratory pressure plateau in pressure-controlled ventilation.

maintained for as long as the patient maintains a spontaneous inspiratory flow. In PCV, however, the pressure plateau is maintained for a preset inspiratory time (Figure 4-10).

PCV is usually indicated for patients with severe ARDS who require extremely high peak inspiratory pressures during mechanical ventilation in a volume-controlled mode. As a result of these high airway pressures, incidence of barotraumas is more likely (Gurevitch et al., 1986).

The advantage of switching these patients from the conventional volume-controlled ventilation to pressure control is that a lower peak inspiratory pressure can be used and maintained while providing oxygenation (PaO_2) and ventilation ($PaCO_2$) (Lain, 1989). Being able to decrease the PIP significantly reduces the risk of barotrauma for these patients.

Table 4-9 summarizes the major characteristics of the pressure-controlled ventilation mode.

TABLE 4-9 Characteristics of the Pressure-Controlled Ventilation Mode

Characteristic	Description
Type of breath	Only mandatory breaths are available to the patient in the pressure-controlled mode.
Triggering mechanism	The mandatory breaths in the pressure-controlled mode are time-triggered by a preset frequency (may be patient-triggered for additional breaths).
Cycling mechanism	The mandatory breaths are time-cycled by a preset inspiratory time.

Notes: The peak inspiratory pressure is controlled by the preset pressure limit. As with any pressure-limited ventilator, the tidal volume will vary directly with lung compliance and inversely with airway resistance. It may be necessary to invert the I:E ratio beyond 1:2 to maintain oxygenation. I:E ratios as high as 4:1 have been reported with successful outcome.

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AIRWAY PRESSURE RELEASE VENTILATION (APRV)

airway pressure release ventilation (APRV): A mode of ventilation in which the spontaneous breaths are at an elevated baseline (i.e., CPAP). This elevated baseline is periodically "released" to facilitate expiration.

Airway pressure release ventilation (APRV) has two CPAP or pressure levels—high pressure (P_{high} or P_{INSP}) and low pressure (P_{low} or PEEP), and the patient is allowed to breathe spontaneously without restriction. When the high pressure (P_{high}) level is dropped or "released" to the low pressure (P_{low}) level, it simulates a mechanical exhalation. Likewise, when the low pressure (P_{low}) level is raised to the high pressure (P_{high}) level, it simulates an inspiratory mechanical breath. In APRV, the patient spends most of the time at the high pressure level with less than 1.5 sec at the low pressure level.

To provide APRV, the ventilator must have a high flow CPAP circuit that has been modified with the addition of a release valve. When the release valve opens, the CPAP pressure is vented and the circuit pressure decreases to zero or a lower CPAP level. Figure 4-11 shows the airway pressure release during CPAP mode.

✓ The tidal volume during APRV is determined by the pressure gradient between CPAP and final pressure following pressure release.

A mandatory inspiration begins with time-triggered closing of the release valve. The airway pressure rapidly increases to the baseline CPAP pressure and is maintained for the duration of inspiration (for as long as the release valve remains closed). The mandatory inspiration ends with time-triggered opening of the release valve, which allows the circuit pressure to decrease as the patient exhales. What is unique about this mode is that the patient is allowed to breathe spontaneously at the high or low pressure levels. Since APRV mode is pressure-limited, for a given pressure gradient ($P_{high} - P_{low}$), the patient's tidal volume will vary directly with changes in lung compliance and inversely with changes in airway resistance. For this reason, the exhaled tidal volume should be closely monitored to prevent hyperinflation.

Patient-ventilator dyssynchrony may result when pressure release (from P_{high} to P_{low}) occurs during spontaneous inspiration, or when pressure increase (from P_{low} to P_{high}) occurs during spontaneous expiration. The timing of pressure release and

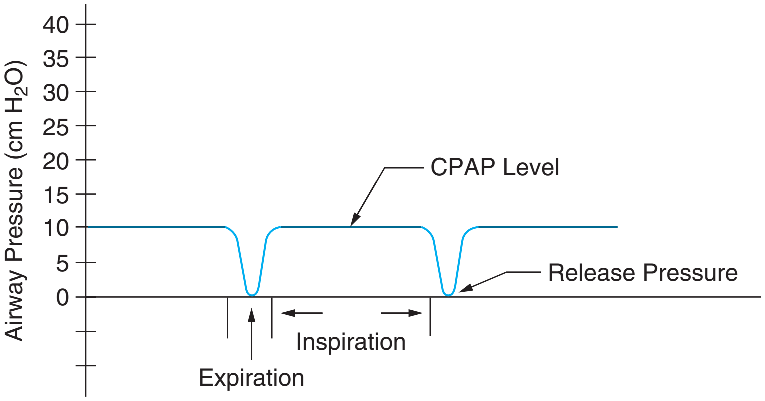

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FIGURE 4-11 Airway pressure release ventilation (APRV) at a CPAP level of 10 cm H_2O and pressure release to 0 cm H_2O with a net release pressure gradient of 10 (10–0) cm H_2O. During APRV, the expiratory phase occurs when the airway pressure is released from 10 to 0 cm H_2O. On inspiration, the CPAP level is maintained at 10 cm H_2O. Since the pressure release time period is rather short, an inversed I:E ratio is usually observed. With APRV, the patient's tidal volume will vary directly with changes in lung compliance and inversely with changes in airway resistance.

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TABLE 4-10 Characteristics of the Airway Pressure Release Ventilation Mode

Characteristic	Description
Type of breath	Time-triggered mandatory breaths will continue in this mode and the patient is allowed to breathe spontaneously between mandatory breaths.
Triggering mechanism	The mandatory breaths are time-triggered, and the patient assumes all spontaneous breaths.
Cycling mechanism	The mandatory breaths are time-cycled by a preset inspiratory time.

pressure increase should be in accordance with the patient's breathing pattern to avoid patient-ventilator dyssychrony.

When bilevel (two-level CPAP) mode on the Puritan Bennett 840 ventilator is in use, the spontaneous breaths have the option of PSV. This PSV feature helps to augment the spontaneous tidal volumes in condition of low compliance and high airflow resistance.

Indications for APRV

The primary indication for this mode is similar to that of pressure control, namely, as an alternative to conventional volume-controlled ventilation for patients with significantly decreased lung compliance such as patients with ARDS. Conventional volume-controlled ventilation in these patients is associated with excessive peak airway pressures and barotrauma. APRV can provide effective partial ventilatory support with lower peak airway pressure than that provided by the PSV and SIMV modes (Chiang et al., 1994). However, APRV may be less comfortable than the PSV and SIMV modes, and synchronization with mechanical breaths may also be a problem.

Table 4-10 summarizes the major characteristics of the airway pressure release ventilation mode.

✓ APRV can provide effective partial ventilatory support with lower peak airway pressure than that provided by the PSV and SIMV modes.

BIPHASIC POSITIVE AIRWAY PRESSURE (Biphasic PAP)

Biphasic positive airway pressure (BiPAP): A mode that has two baseline pressures (P_{insp} and PEEP). It allows spontaneous breathing at any point in the mechanical ventilation cycle.

Biphasic positive airway pressure (biphasic PAP or **BiPAP**) is a mode that has two baseline pressure levels (P_{INSP} and PEEP) and it allows spontaneous breathing at any point in the mechanical ventilation cycle. Biphasic PAP is also called Bilevel (Puritan Bennett), BIPAP (Drager Europe), Bi-Vent (Siemens), BiPhasic (Avea), PCV+ (Drager Medical), and DuoPAP (Hamilton) (Mireles-Cabodevila et al., 2009).

Biphasic PAP is similar to APRV with one exception. In APRV, the patient spends most of the time at the high pressure level with less than 1.5 sec at the low pressure level. In Biphasic PAP, the patient spends more time at the low pressure level (Mireles-Cabodevila et al., 2009). Figure 4-12 shows the duration of time at high and low pressure levels for APRV and Biphasic PAP.

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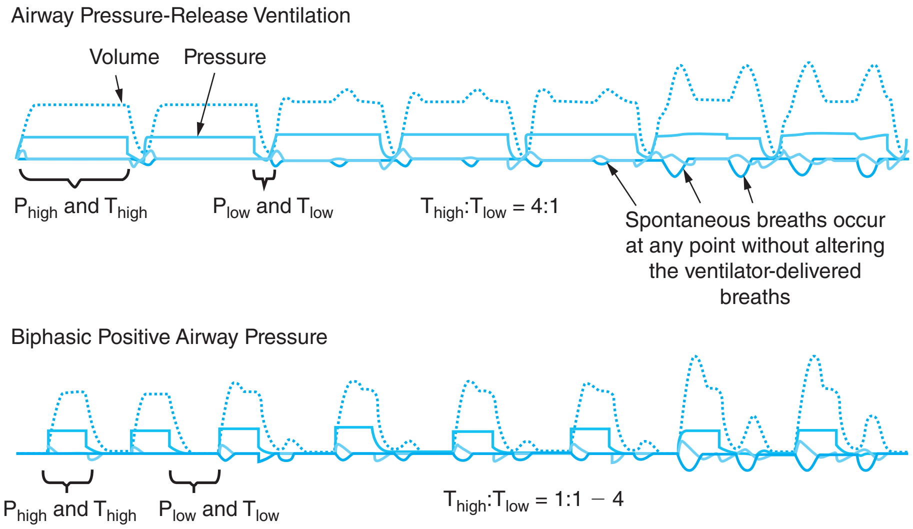

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FIGURE 4-12 APRV and BiphasicPAP waveforms. In biphasicPAP, the patient spends more time at the low pressure level. (Reference: Mireles-Cabodevila, 2009)

INVERSE RATIO VENTILATION (IRV)

The ratio of inspiratory time (I time) to expiratory time (E time) is known as the I:E ratio. In conventional mechanical ventilation, the I time is traditionally lower than the E time so that the I:E ratio ranges from about 1:1.5 to 1:3. This resembles the normal I:E ratio during spontaneous breathing, and it is considered physiologically beneficial to normal cardiopulmonary function.

Since the mid-1980s, investigators have been extending the inspiratory time during mechanical ventilation to promote oxygenation in patients with ARDS (Gurevitch et al., 1986; Marcy et al., 1991). The inverse I:E ratio in use is between 2:1 and 4:1 and often it is used in conjunction with pressure-controlled ventilation (Lain et al., 1989; Tharratt et al., 1988).

Physiology of IRV

✓ Inverse ratio ventilation (IRV) improves oxygenation by (1) ↓ intrapulmonary shunting, (2) ↑ V/Q matching, and (3) ↓ deadspace ventilation.

Inverse ratio ventilation (IRV) improves oxygenation by (1) reduction of intrapulmonary shunting, (2) improvement of V/Q matching, and (3) decrease of deadspace ventilation. From the review of available literature, Shanholtz et al. et al. (1994) concluded that these mechanisms were also achievable by use of conventional ventilation with PEEP. However, two notable changes are observed during IRV. They are (1) increase of mean airway pressure and (2) presence of auto-PEEP. These two changes are likely the reason for the improvement of shunting and hypoxemia in ARDS patients.

✓ The increase in mPaw during IRV helps to reduce shunting and improve oxygenation in ARDS patients.

Increase of Mean Airway Pressure. To achieve the same degree of ventilation and oxygenation, IRV requires a lower peak airway pressure and PEEP, but a higher mean airway pressure (mPaw) than conventional mechanical ventilation. The increase in mPaw during IRV helps to reduce shunting and improve oxygenation in ARDS patients (Shanholtz et al., 1994).

✓ The presence of auto-PEEP during IRV may help to reduce shunting and improve oxygenation in ARDS patients.

Addition of Auto-PEEP. Since IRV provides a longer I time and shorter E time, breath stacking with an increase of end-expiratory pressure is likely when there is not enough time for complete expiration (Duncan et al., 1987; Kacmarek et al., 1990). The presence of auto-PEEP during IRV may help to reduce shunting and improve oxygenation in ARDS patients (Shanholtz et al., 1994).

Adverse Effects of IRV

During IRV, the increase in mPaw and the presence of auto-PEEP both contribute to the increase of mean alveolar pressure and volume, and the incidence of barotrauma may be as high as that obtained by conventional ventilation with high levels of PEEP (Tharratt et al., 1988).

Another potential hazard of IRV is a higher rate of transvascular fluid flow or flooding induced by an increased alveolar pressure (Permutt, 1979). This condition may induce or worsen preexisting pulmonary edema.

Patients receiving IRV are often agitated. They may require sedation and neuromuscular blocking agents to facilitate ventilation. The associated complications with these drugs can be serious and they should be monitored carefully when used in conjunction with IRV (Hansen-Flaschen et al., 1993).

Pressure Control-IRV (PC-IRV)

✓ When PC is used with IRV, the peak airway pressure may be kept at a safe level.

Since IRV may increase the mPaw, create auto-PEEP, and increase the incidence of barotrauma, it is sometimes used in conjunction with pressure-controlled ventilation due to its pressure-limiting capability. By using pressure control, the peak airway pressure may be kept at a safe level. This strategy helps to minimize pressure-induced lung injuries. When an inverse I:E ratio is used with pressure-controlled ventilation, it is called pressure control-inverse ratio ventilation (PC-IRV).

Several studies compare the outcomes of ARDS patients before and after the implementation of PC-IRV. The changes that may occur when positive pressure ventilation with PEEP (PPV + PEEP) is switched over to the PC-IRV mode of ventilation are summarized in Table 4-11.

TABLE 4-11 Observed Changes after Switching from PPV + PEEP to PC-IRV

Increase	Decrease	No Change
Mean airway pressure	PEEP requirement	F_IO_2 requirement
Central venous pressure	Peak airway pressure	Intrinsic PEEP
Pulmonary artery pressure	Cardiac output	Blood pressure
PaO_2		$PaCO_2$

(Data from East et al., 1992; Lain et al., 1989, 1990.)

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AUTOMATIC TUBE COMPENSATION (ATC)

automatic tube compensation (ATC): A mode of ventilation that offsets and compensates for the airflow resistance imposed by the artificial airway.

Automatic tube compensation (ATC) is available in the Evita 4 ventilator (Dräger Medical). The PB840 ventilator has a similar feature which is called tubing compensation (TC). This tubing compensation can be applied in all ventilation modes. ATC offsets and compensates for the airflow resistance imposed by the artificial airway. It allows the patient to have a breathing pattern as if breathing spontaneously without an artificial airway. With ATC, the pressure delivered by the ventilator to compensate for the airflow resistance is active during inspiration and expiration. It is dependent on the airflow characteristics and the flow demand of the patient. For example, when the airway diameter decreases or flow demand increases, the pressure is raised to overcome a higher airflow resistance or increased flow demand.

NEURALLY ADJUSTED VENTILATORY ASSIST (NAVA)

Neurally adjusted ventilatory assist (NAVA): A mode of mechanical ventilation in which the patient's electrical activity of the diaphragm (EAdi or Edi) guides the optimal functions of the ventilator.

Neurally adjusted ventilatory assist (NAVA) is a mode of mechanical ventilation in which the patient's electrical activity of the diaphragm (EAdi or Edi) is used to guide the optimal functions of the ventilator (Spahija et al., 2005). The neural controls of respiration originated in the patient's respiratory center are sent to the diaphragm via the phrenic nerves. In turn, bipolar electrodes are used to pick up the electrical activity. The electrodes are mounted on a disposable EAdi catheter and positioned in the esophagus at the level of the diaphragm (Maquet, 2009).

NAVA is available for adults, children, and neonates, and it has been used successfully in the management and weaning of mechanically ventilated patients with spinal cord injury. Other uses and potential applications of NAVA include patients with head injury, COPD, and history of ventilator dependency (Maquet, 2011). The ability to wean these patients rapidly reduces or eliminates the incidence of disuse atrophy of the diaphragm (Betters et al., 2004).

HIGH-FREQUENCY OSCILLATORY VENTILATION (HFOV)

High-frequency oscillatory ventilation (HFOV): HFOV does not have a tidal volume setting and it delivers extremely small volumes at high frequency,

High-frequency oscillatory ventilation (HFOV) delivers extremely small volumes at high frequency. Its main application is to minimize development of lung injury while providing mechanical ventilation. HFOV was patented in 1952 by Emerson and was developed for clinical application in the early 1970s by Lunkenheimer. The U.S. Food and Drug Administration approved HFOV for clinical use in neonates in 1991, children in 1995, and adults in 2001. The primary settings of HFOV ventilators are: Airway pressure amplitude (delta P or power), frequency, mean airway pressure, percent inspiration, inspiratory bias flow, and F_IO_2.

✔ In HFOV, ventilation can be increased by *decreasing* the oscillation frequency. Ventilation can also be increased by increasing the amplitude of the oscillations, inspiratory time, or bias flow (with an intentional cuff leak).

✔ In HFOV, oxygenation to the patient can be increased by increasing the mean airway pressure or increasing the F_IO_2.

HFOV delivers a constant flow (bias flow) and its piston pump oscillates at frequencies ranging from 3 Hz to 15 Hz (180 breaths/min to 900 breaths/min). Adult patients are sedated to prevent deep spontaneous breathing, as this will trigger the alarms and affect ventilator performance.

Ventilation can be increased by *decreasing* the oscillation frequency (in traditional ventilators, the frequency would be increased). Ventilation can also be increased by increasing the amplitude of the oscillations, increasing the inspiratory time, or increasing the bias flow (with an intentional cuff leak). Oxygenation to the patient can be increased by increasing the mean airway pressure or increasing the F_IO_2. Topics related to high frequency ventilation will be discussed in more details in Chapter 17, "Neonatal Mechanical Ventilation."

SUMMARY

There are many different ventilator operating modes and the number is expected to increase in coming years. As each mode is designed to accomplish a set of specific functions, it is essential to understand its capabilities, as well as its limitations and complications. When two or more operating modes are used in tandem, care and caution must be used because the combined outcomes are often complex and difficult to predict or manipulate.

An excellent source of obtaining detailed technical information is to consult the operation manual or contact the technical professionals of each ventilator manufacturer. Quality patient care is possible when the appropriate operating modes are selected and applied. A willingness to participate in continuing education is highly desirable and sometimes mandatory in this ever-changing field of mechanical ventilation.

Self-Assessment Questions

1. Volume-controlled ventilation delivers a predetermined ________ to the patient using variable ________ according to the changing compliance and resistance.
   - A. tidal volume, peak inspiratory pressure
   - B. peak airway pressure, tidal volume pressure
   - C. tidal volume, PEEP
   - D. PEEP, pressure support

2. During pressure-controlled ventilation, an increase of airway resistance or a decrease of compliance would:
   - A. reduce the delivered volume.
   - B. raise the peak inspiratory pressure.
   - C. reduce the work of breathing.
   - D. cause respiratory alkalosis.

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3. Positive end-expiratory pressure (PEEP) is most commonly used to correct:

   A. hypercapnia.
   B. mild hypoxemia.
   C. refractory hypoxemia.
   D. respiratory acidosis.

4. When PEEP is applied to the airway of a spontaneously breathing patient, it is called:

   A. airway pressure release ventilation.
   B. continuous positive airway pressure.
   C. pressure support ventilation.
   D. pressure-controlled ventilation.

5. _______ is a mode in which the ventilator delivers control (mandatory) breaths and allows the patient to breathe spontaneously between the mandatory breaths.

   A. Intermittent mandatory ventilation
   B. Continuous positive airway pressure
   C. Mandatory minute ventilation
   D. Airway pressure release ventilation

6. _______ delivers control (mandatory) breaths to the patient at or near the beginning of a spontaneous breath, thus avoiding breath stacking.

   A. Continuous positive airway pressure
   B. Airway pressure release ventilation
   C. Intermittent mandatory ventilation
   D. Synchronized intermittent mandatory ventilation

7. _______ best describes the mode of ventilation that changes the number of mandatory breaths and pressure support level according to the patient's breathing pattern.

   A. Automatic tube compensation
   B. Volume ventilation plus
   C. Adaptive support ventilation
   D. Volume-assured pressure support

8. _______ is primarily used to reduce the work of breathing imposed by the endotracheal tube and ventilator circuit during spontaneous breathing.

   A. Pressure support ventilation
   B. Pressure-controlled ventilation
   C. Airway pressure release ventilation
   D. Continuous positive airway pressure

9. _______ is a mode of ventilation that changes the pressure support level according to the changes in volume, elastance, airflow resistance, and flow demand.

   A. Proportional assist ventilation (proportional pressure support)
   B. Pressure support ventilation
   C. Volume-assured pressure support
   D. Automatic tube compensation

10. _______ is a mode of ventilation that assures a stable tidal volume by using pressure support ventilation and volume-assisted ventilation.

    A. Automatic tube compensation
    B. Pressure support ventilation
    C. Proportional pressure support
    D. Volume-assured pressure support

11. In pressure-regulated volume control, the peak inspiratory pressure is kept at a lowest level possible by altering the _______ in response to changing airway or compliance characteristics.

    A. peak flow and tidal volume
    B. peak flow and inspiratory time
    C. plateau pressure and tidal volume
    D. plateau pressure and inspiratory time

12. Volume ventilation plus (VV+) provides the following options:

    A. volume control plus, and pressure support.
    B. volume control plus and volume support.
    C. volume-assured pressure support and pressure support.
    D. volume-assured pressure and support volume support.

13. When the patient has no spontaneous breathing efforts, pressure-controlled ventilation delivers _______ -triggered breaths and a _______ is created and maintained for a preset inspiratory time.

    A. pressure, PEEP
    B. time, pressure plateau
    C. flow, pressure plateau
    D. patient, PEEP

14. One distinctive characteristic of airway pressure release ventilation is that exhalation occurs when the:

    A. low pressure is changed to high pressure.
    B. low pressure is changed to 0 cm H_2O.
    C. high pressure is changed to low pressure.
    D. high pressure is changed to 0 cm H_2O.

15. Inverse ratio ventilation has been used successfully to reduce intrapulmonary shunting and improve oxygenation. These effects are likely the result of _______ and _______.

    A. deadspace ventilation, increase of peak inspiratory pressure
    B. deadspace ventilation, auto-PEEP
    C. increase of mean airway pressure, auto-PEEP
    D. increase of peak inspiratory pressure, auto-PEEP

16. In high-frequency oscillatory ventilation, hypoventilation should be managed by decreasing the:

    A. inspiratory time.
    B. bias flow.
    C. amplitude.
    D. frequency.

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Answers to Self-Assessment Questions

1. A.
2. A.
3. C.
4. B.
5. A.
6. D.
7. C.
8. A.
9. A.
10. D
11. B.
12. B.
13. B.
14. C.
15. C.
16. D.

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Chapter 5

Special Airways For Ventilation

David W. Chang

Outline

- Introduction
- Oropharyngeal Airway
  - *Types of Oropharyngeal Airway*
  - *Selection of Oropharyngeal Airway*
  - *Insertion of Oropharyngeal Airway*
- Nasopharyngeal Airway
  - *Selection of Nasopharyngeal Airway*
  - *Insertion of Nasopharyngeal Airway*
  - *Complications of Nasopharyngeal Airway*
- Esophageal Obturator Airway (EOA)
  - *Insertion of EOA*
- Esophageal Gastric Tube Airway (EGTA)
- Laryngeal Mask Airway (LMA)
  - *Use of LMA*
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- Esophageal-Tracheal Combitube (ETC)
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  - *Indications*
  - *Selection of DLT*
  - *Insertion of DLT*
  - *Complications of DLT*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

- autoclave
- blind distal end
- blind intubation
- double-lumen endobronchial tube
- esophageal gastric tube airway (EGTA)
- esophageal obturator airway (EOA)
- esophageal-tracheal combitube (ETC)
- laryngeal mask airway (LMA)
- oropharyngeal airway

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Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Name the types of oropharyngeal airways and describe the methods to select and insert these airways.
- Describe the characteristics of an esophageal obturator airway and the procedure to insert this airway.
- List the clinical uses and limitations of a laryngeal mask airway and describe how to select, insert, and remove this airway.
- List the clinical uses of an esophageal-tracheal combitube and the potential complications of this airway.
- List the clinical uses of a double-lumen endobronchial tube and the method to select and insert this airway.

INTRODUCTION

In situations involving respiratory arrest, bag and mask ventilation is typically used and may be followed by endotracheal intubation. Occasionally, endotracheal intubation may not be successful due to unusual anatomy or difficult clinical setting. In these cases, a special airway, such as the esophageal obturator airway, esophageal gastric tube airway, laryngeal mask airway, or esophageal-tracheal combitube (ETC), may serve as a stopgap measure for providing ventilation when bag and mask ventilation is deemed inadequate. The double-lumen endobronchial tube is another special airway for conditions where independent lung ventilation is indicated.

OROPHARYNGEAL AIRWAY

oropharyngeal airway: A device to relieve upper airway obstruction.

✓ An oropharyngeal airway should be used in patients who are sedated or unconscious.

An **oropharyngeal airway** is used to relieve upper airway obstruction if airway maneuvers (e.g., head tilt-chin lift, jaw thrust) fail to open an unobstructed airway (White, 2004). During bag-mask ventilation, an oropharyngeal airway may facilitate effective ventilation. It may also be used as a bite block in intubated patients. An oropharyngeal airway should be used in patients who are sedated or unconscious. For conscious patients, insertion of this airway may trigger the gag reflex and cause vomiting, and aspiration of stomach contents into the lungs.

Types of Oropharyngeal Airways

There are two major types of oropharyngeal airways (Figure 5-1). The Berman airway has external side channels and ranges from size 43 mm for infants to size

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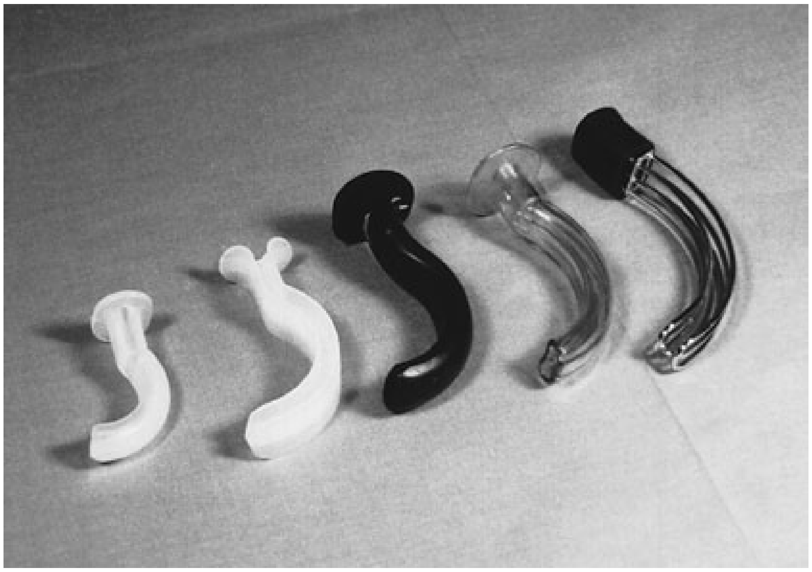
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FIGURE 5-1 A variety of oropharyngeal airways. Berman airways, a Guedel airway, and a Cath-Guide Guedel airway are shown.

TABLE 5-1 Size Chart for Berman and Guedel Oropharyngeal Airways

	Extra-Large Adult	Large Adult	Medium Adult	Small Adult	Child	Infant
Berman	110 mm	100 mm	90 mm	80 mm	60 mm	43 mm
Guedel	120 mm	110 mm	100 mm	80 mm	60, 70 mm	55 mm

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110 mm for extra-large adults. The common Guedel airway has one large internal channel and the Cath-Guide Guedel has three internal channels. Guedel airways have sizes ranging from 55 mm for infants to 120 mm for extra-large adults. (See Table 5-1.)

Selection of Oropharyngeal Airway

✓ The appropriate size of an oropharyngeal airway may be estimated by the distance from the center of the mouth (or central incisors) to the angle of the jaw or from the corner of the mouth to the earlobe.

The appropriate size (from flange to distal tip) of an oropharyngeal airway may be estimated based on the length in millimeters from the center of the mouth to the angle of the jaw. Alternatively, the length in millimeters from the corner of the mouth to the earlobe may be used. The third method is to measure the distance from the central incisors to the angle of the jaw. To evaluate the size using this method, place the airway next to the patient's face.

Proper sizing for the patient is important. If the airway is too large, it may push the epiglottis against the larynx leading to airway obstruction. If the airway is too small, the tongue may not be sufficiently moved away from the soft palate leading to airway obstruction by the tongue.

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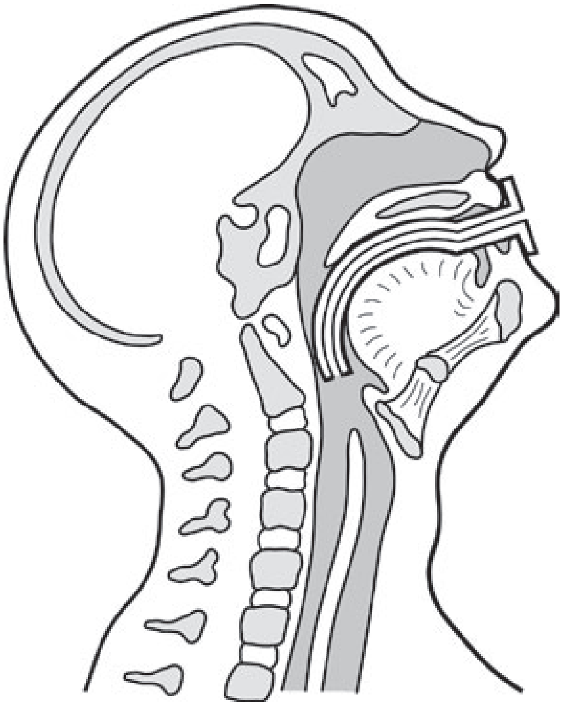
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FIGURE 5-2 The correct placement of an oropharyngeal airway. Note how the distal tip of the airway rests at the base of the tongue.

Insertion of Oropharyngeal Airway

Prior to insertion of an oropharyngeal airway, ensure that the patient is sedated or unconscious. If the patient begins to gag or retch during the procedure, remove the airway immediately and reassess the necessity of an oropharyngeal airway. Sometimes the airway may be opened and maintained by repositioning of the head (e.g., head tilt-chin lift, jaw thrust).

Body fluid or isolation precautions must be observed throughout the procedure. The patient should be in a supine position, and the mouth is opened using the scissors (crosses fingers) technique. If a tongue blade is available, the tongue is depressed and the oropharyngeal airway may be inserted with the pharyngeal curvature. Some practitioners prefer to insert the airway into the patient's mouth upside down so that the distal end of the airway is facing the hard palate (roof of the patient's mouth). As the airway is inserted fully, it is turned 180° until the flange (proximal end) rests on the patient's lips or teeth (White, 2004). The correct placement of an oropharyngeal airway is shown in Figure 5-2.

NASOPHARYNGEAL AIRWAY

The nasopharyngeal airway is a simple airway adjunct that can be used to facilitate ventilation and removal of secretions. It is also called a nasal trumpet or nasal horn (White, 2013). A nasopharyngeal airway has advantages over the oropharyngeal airway

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TABLE 5-2 Size Chart for Nasopharyngeal Airways

Patient	Average Size
Short female	6 (pin 1 cm from flange)
Average female/short male	6
Tall female/average male	7
Tall male	8

as this nasal airway can be used in patients with an intact gag reflex. Other indications include patients with unstable fractures of the mandible, trimus (lockjaw), or oral trauma (Roberts et al., 2005).

✓ An nasopharyngeal airway can be used in patients with an intact gag reflex, unstable fractures of the mandible, trimus (lockjaw), or oral trauma

Selection of Nasopharyngeal Airway

The appropriate size of nasopharyngeal airway for average females is a size 6. For average males, the size should be a size 7. The final selection should be based on the patient's height and clinical condition. The methods of using the width of the patient's nares or size of the little finger are based on anecdotal teaching rather than clinical evidence (Roberts et al., 2003).

Proper sizing for the patient is important. If the nasopharyngeal airway is too short, the airway would not separate the soft palate from the posterior wall of the pharynx. If the airway is too long, it would enter either the larynx and aggravate laryngeal reflexes or enter the space between the epiglottis and the vallecula leading to potential obstruction of the airway. The ideal length of the nasopharyngeal airway should have the distal end of the airway within 1 cm of the epiglottis (Stoneham, 1993). Table 5-2 shows the size chart for nasopharyngeal airways.

Insertion of Nasopharyngeal Airway

Prior to insertion of a nasopharyngeal airway, the nares should be inspected for obstruction. A local anesthetic spray may be applied to the posterior nares for patient comfort. Prior to insertion, the patient should be in a sitting or semi-Fowler position and the nares are lifted to reveal the nasal airway. Placement of the airway should be parallel to the nasal floor, rather than upwards toward the cribriform plate of the ethmoid bone. Lubrication with a water-soluble lubricant and gentle rotation should facilitate the insertion (Roberts et al., 2005). Body fluid or isolation precautions must be observed throughout the procedure. A nasopharyngeal airway and its correct placement are shown in Figures 5-3 and 5-4.

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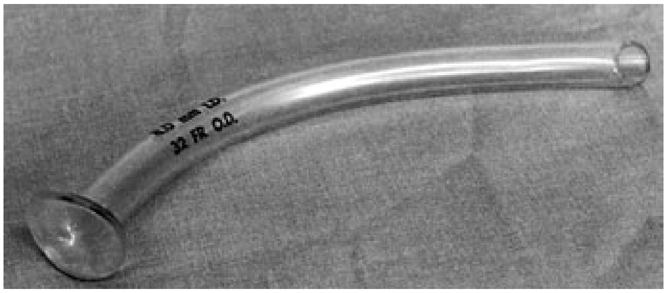

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FIGURE 5-3 A nasopharyngeal airway.

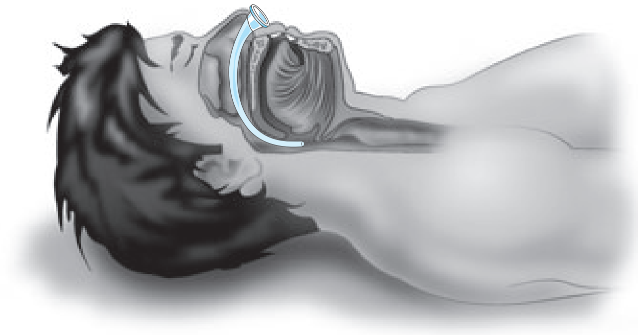

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FIGURE 5-4 Proper placement of a nasopharyngeal airway.

Complications of Nasopharyngeal Airway

The nasopharyngeal airway is unstable and it should be inspected for inadvertent movements. Outward movement is more common. Inward migration may be prevented by using a safety pin on the distal end of the airway to prevent it from going into the nares. Other common complications include soft tissue damage of the nasal mucosa and bleeding. There are two reported cases that involved basilar skull fracture with use of nasopharyngeal airway (Roberts et al., 2005).

ESOPHAGEAL OBTURATOR AIRWAY (EOA)

esophageal obturator airway (EOA): An EOA has a closed (blind) distal end and it is inserted into the esophagus.

blind distal end: The far end of a tube without an opening.

Unlike an endotracheal tube, an **esophageal obturator airway (EOA)** is inserted into the esophagus. It is used as an alternative to bag and mask ventilation. The EOA is a disposable tube; its structure consists of an opening at the proximal (top) end, many small holes near the mid-section, and a **blind distal end**. Near the distal end is a large cuff that is inflated during use. The inflated cuff prevents air from entering the stomach and subsequent regurgitation and aspiration. A mask fits over the tube to prevent leaks around the patient's

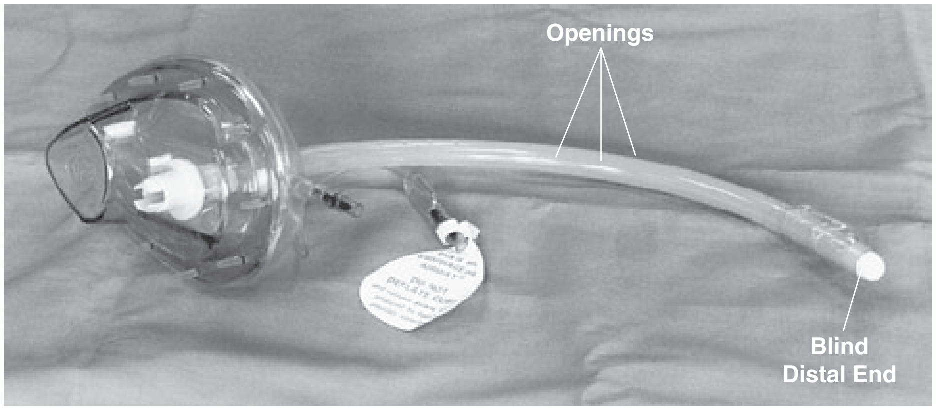

FIGURE 5-5 An esophageal obturator airway (EOA).

face during ventilation (Burton et al., 1997; White, 2004). Figure 5-5 shows an esophageal obturator airway.

The opening at the proximal end of the tube attaches to a ventilation bag. The small holes at the hypopharyngeal level allow ventilation to the lungs. The closed distal end of the EOA prevents aspiration or removal of air or gastric contents from the stomach. Since an EOA is inserted into the esophagus, the cuff at the distal end must be inflated during use to prevent air from entering the stomach (Wilkins et al., 2003; White, 2004).

✓ Since an EOA is inserted into the esophagus, the cuff at the distal end must be inflated during use to prevent air from entering the stomach.

Insertion of EOA

The cuff of an EOA is first inflated with 20 to 30 mL of air to check for cuff integrity and leaks. The cuff is then deflated, and the proximal end of the EOA is inserted through the opening of a mask. The distal end of the tube is lubricated with a water-soluble lubricant and then inserted into the patient's esophagus until the mask rests on the patient's face. Due to the large volume of air used to inflate the cuff, it is extremely important to check for proper tube placement before cuff inflation and ventilation. Asphyxia and tracheal damage are severe complications if the cuff is inflated while the tube is misplaced in the trachea (Wilkins et al., 2003; White, 2004). Table 5-3 lists other precautions during use of the EOA.

The EOA is not designed to be used as an artificial airway for positive pressure ventilation. Since it is used as a temporary airway, it should be replaced with an endotracheal intubation as soon as feasible. With the EOA in place, endotracheal intubation is done using the standard procedure. After endotracheal intubation, bilateral breath sounds should be verified as the endotracheal tube may follow the EOA and enter the esophagus. After ascertaining correct placement, the endotracheal tube is secured prior to removal of the EOA. Suction setup should be ready in case of vomiting during removal of the EOA.

✓ The EOA is not designed to be used as an artificial airway for positive pressure ventilation.

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TABLE 5-3 Precautions in the Use of an Esophageal Obturator Airway
An EOA should not be used in awake or semiconscious patients.
An EOA should not be used in children under 16 years of age or under 5 ft tall.
An EOA should not be used in patients with known esophageal disease.
An EOA must not be removed until the patient has regained consciousness.

ESOPHAGEAL GASTRIC TUBE AIRWAY (EGTA)

esophageal gastric tube airway (EGTA): A tube used in esophageal intubation. It has a patent distal end to relieve gastric distention.

✓ There are two ports on the EGTA mask. The resuscitation bag must be attached to the ventilation port.

The **esophageal gastric tube airway (EGTA)** is similar in design to the EOA. Whereas the EOA has a closed distal end, the EGTA has an opening at the distal end (Figure 5-6). The opening allows removal or aspiration of air and gastric contents from the stomach via a gastric tube. The advantage of this design is the relief of gastric distention or pressure that may occur during bag to mask ventilation (Wilkins et al., 2003; White, 2004).

With an EGTA, the ventilation holes along the proximal end of the tube are absent. Ventilation is provided through the mask by a traditional manual resuscitation bag. Since there are two ports on the mask, the resuscitation bag must be attached to the ventilation port. Table 5-4 outlines the distinct features of the EOA and the EGTA.

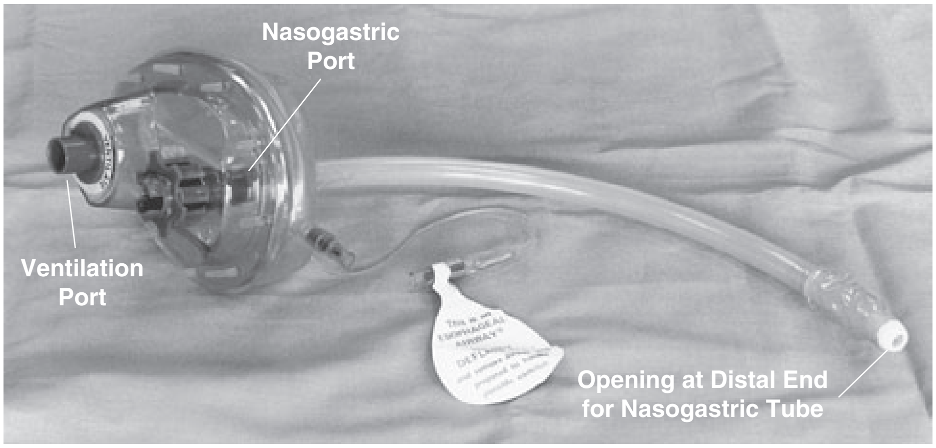

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FIGURE 5-6 An esophageal gastric tube airway (EGTA).

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TABLE 5-4 Distinct Features of the EOA and the EGTA

Esophageal Obturator Airway	Esophageal Gastric Tube Airway
Esophageal intubation	Esophageal intubation
Ventilation holes along tube	No ventilation holes along tube
Blind distal end	Patent distal end
One port on mask for ventilation	Two ports on mask (ventilation/gastric tube)

LARYNGEAL MASK AIRWAY (LMA)

laryngeal mask airway (LMA): A tube with a small cushioned mask on the distal end that provides a seal over the laryngeal opening.

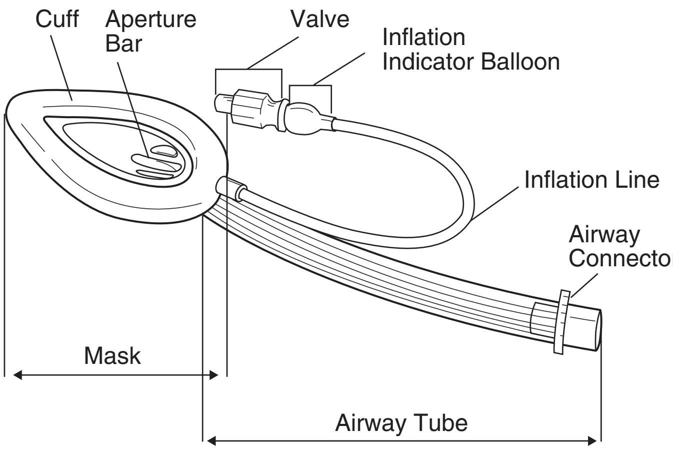
✓ The original LMA is reusable.

✓ The cushioned mask of the LMA provides a seal over the laryngeal opening. It is not necessary for the LMA to enter the larynx or trachea.

The **laryngeal mask airway (LMA)** resembles a short endotracheal tube with a small cushioned, oblong-shaped mask on the distal end (Figure 5-7) (Brain et al., 1997; Brimacombe et al., 1998; Verghese et al., 1998). It was invented in England in 1981 by anesthesiologist Archie Brain and was available commercially in 1988. In 1991, this airway device was approved by the Food and Drug Administration for clinical use in the United States (Ferson et al., 1997). The original LMA (LMA-Classic) is a reusable device, made primarily of medical-grade silicone rubber and is latex-free. With proper care and sterilization, it can be reused up to 40 times.

Use of LMA

The LMA fills a niche as an airway management tool between a face mask and an endotracheal tube (Brimacombe et al. 1996; Fetzer, 1998). When the cushioned mask of the LMA is inflated, it provides a seal over the laryngeal opening. It is not



Courtesy of LMA North America, Inc.

FIGURE 5-7 The components of the laryngeal mask airway (LMA).

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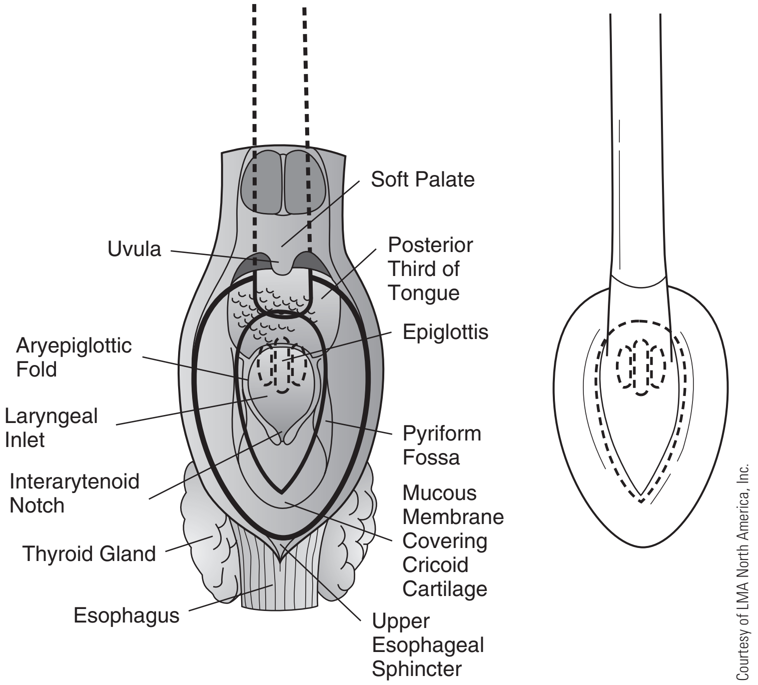

Courtesy of LMA North America, Inc.

FIGURE 5-8 Dorsal view of the LMA showing position in relation to pharyneal anatomy.

necessary therefore for the LMA to enter the larynx or trachea. Figure 5-8 shows the dorsal view of the position of the LMA in relation to the pharyngeal anatomy. After proper placement, spontaneous ventilation or low-level positive pressure ventilation (up to 20 cm H_2O for most LMA types) is possible without an endotracheal tube (LMA North America, Inc., 1999).

The LMA is also indicated as a method of establishing a patent airway during resuscitation in the profoundly unconscious patient with absent glossopharyngeal and laryngeal reflexes who may need assisted ventilation. The LMA should be considered when tracheal intubation is precluded by lack of expertise or equipment or when attempts at endotracheal intubation have failed (LMA North America, Inc., 1999). According to the adult Basic Life Support (BLS), LMA is recommended as an alternative airway to the bag-mask device. In adult Advanced Cardiovascular Life Support (ACLS), the LMA is classified as an intervention that is acceptable, safe, and useful. In Neonatal Resuscitation, the LMA is recommended as an alternative in a situation of "cannot intubate" or "cannot ventilate" (LMA North America, Inc., 2012).

✓ LMA should be considered when tracheal intubation is precluded by lack of expertise or equipment or when attempts at endotracheal intubation have failed.

Other uses and application of the LMA are outlined in Table 5-5.

Contraindications for LMA

The design of the LMA does not protect an airway from the effects of regurgitation and aspiration. For this reason, the LMA should not be used in patients who have not fasted or those with hiatal hernia. The LMA should not be used in patients who are not profoundly unconscious and in those with severe oropharyngeal trauma.

✓ The LMA does not protect an airway from the effects of regurgitation and aspiration.

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TABLE 5-5 Uses and Application of a Laryngeal Mask Airway
Establishes airway in proven difficult intubations
Provides spontaneous and controlled ventilation in infants and children
Serves as a bridge to more secured airways
Provides complete survey of the larynx and trachea prior to thoracotomy
Provides lower work of breathing than endotracheal tube
Provides less hemodynamic response during surgical procedures
Provides less airway reaction
Offers benefit of shorter stay in hospital due to avoidance of endotracheal intubation

(Data from Ferson et al., 1997; Fukutome et al., 1998; Joo & Rose, 1998; Joshi et al., 1998; Kim & Bishop, 1999; Lopez-Gil, Brimacombe & Alvarez, 1996; Marietta et al., 1998; Parmet et al., 1998; Stanwood, 1997; Webster et al., 1999; Zerafa et al., 1999.)

The LMA should not be used as a conduit for emergency resuscitation drugs (e.g., epinephrine) because of low success rate (27% in one study). It may be used as an option in emergency situations where a venous access or an endotracheal tube is not readily available (Alexander et al., 1997; Challiner et al., 1997).

Selection of LMA

For most adults, size 4 should be used for females and size 5 for males.

A larger LMA with less air in cuff provides a better seal.

The LMA is reusable (silicone-based) or disposable (polyvinyl chloride). The disposable LMA-Unique performs similarly to the reusable LMA in clinical situations (Verghese et al., 1998). For most adult females, size 4 should be used, and size 5 should be used for most adult males (Asai et al., 1998). A larger LMA with less air in cuff provides a better seal. A smaller LMA along with overinflation of the cuff reduces the cuff compliance, resulting in an improper fit within the pharyngeal space. When the maximum cuff volume is exceeded, air leak, gastric insufflations, and mask malposition become more likely (Brimacombe & Brain, 1997; Ferson et al., 1998).

The standard cuff pressure is 60 cm H_2O (Berry et al., 1998), but the air in the cuff should be adjusted to the minimal effective volume so as to decrease intracuff pressure, pressure on the pharynx (Asai et al., 1998), and incidence of sore throat (Nott, 1998). Table 5-6 provides the suggested LMA size for patients ranging from neonates to large adults and the maximal cuff inflation volume (LMA North America, Inc., 1999).

Insertion of LMA

Different LMA types or brands require different insertion techniques. Users must follow the manufacturer's guidelines or recommendations for the insertion of LMA. Prior to insertion of the LMA, the patient is in a supine position, and the head is advanced slightly. The chin is depressed to open the mouth. With the cuff

TABLE 5-6 Selection of Laryngeal Mask Airway and Maximum Cuff Inflation Volume

Size	Patient Group	Maximum Cuff Volume
1	Neonates and infants up to 5 kg	4 mL
1.5	Infants between 5 and 10 kg	7 mL
2	Infants and children between 10 and 20 kg	10 mL
2.5	Children between 20 and 30 kg	14 mL
3	Children between 30 to 50 kg and small adults	20 mL
4	Adults 50 to 70 kg	30 mL
5	Adults 70 to 100 kg	40 mL
6	Adults over 100 kg	50 mL

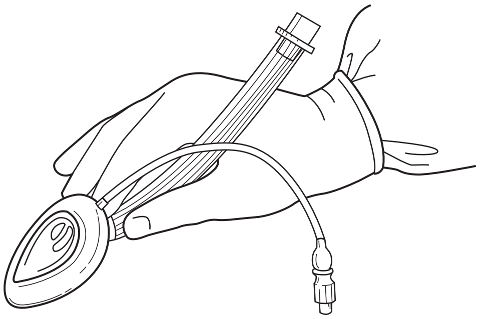
(Data from LMA North America, Inc., 2012.)

completely deflated or partially inflated (Dingley & Asai, 1996), the LMA is inserted blindly without a laryngoscope through the mouth and advanced along the hard palate. It is then further advanced to the posterior pharynx and turned toward the trachea and larynx. At this point, the LMA may be guided with fingers to ascertain that it makes the proper turn (Watson et al., 1999). Figure 5-9(A)–(G) show the standard insertion technique of the LMA.

Removal of LMA

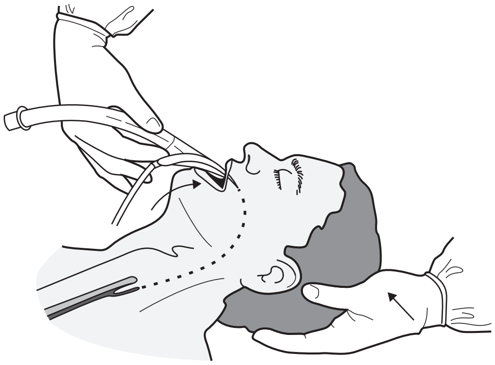
✓ The LMA may be removed safely when the patient is anesthetized or awake.

The LMA may be discontinued when an upper airway is no longer needed for ventilation and oxygenation. Removal can be done safely when the patient is anesthetized or awake. During removal of the LMA, the patient must be monitored carefully



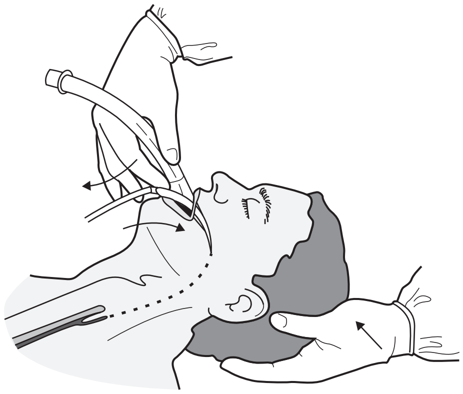
Courtesy of LMA North America, Inc.

FIGURE 5-9(A) Method for holding the LMA for insertion.



Courtesy of LMA North America, Inc.

FIGURE 5-9(B) With the head extended and the neck flexed, carefully flatten the LMA tip against the hard palate.



Courtesy of LMA North America, Inc.

FIGURE 5-9(C) To facilitate LMA introduction into the oral cavity, gently press the middle finger down on the jaw.

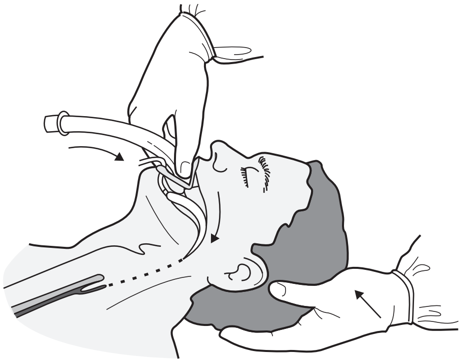


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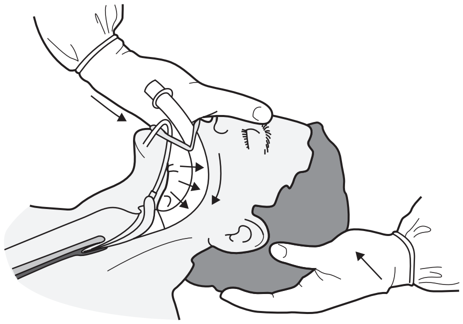
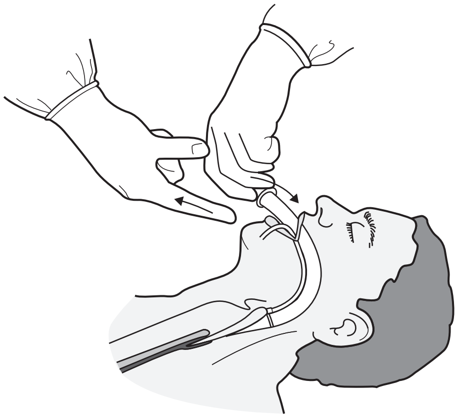
FIGURE 5-9(D) The index finger pushes the LMA in a cranial direction following the contours of the hard and soft palate.



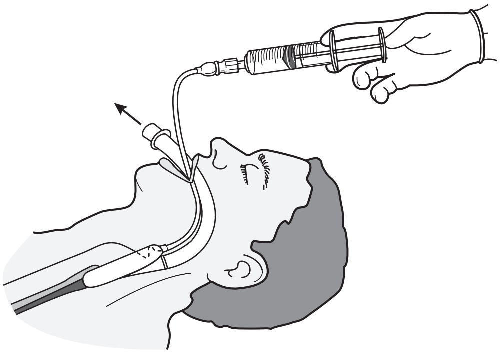
Courtesy of LMA North America, Inc.

FIGURE 5-9(E) Maintaining pressure with the finger on the tube in the cranial direction, advance the mask until definite resistance is felt at the base of the hypopharynx.



Courtesy of LMA North America, Inc.

FIGURE 5-9(F) Gently maintain cranial pressure with the nondominant hand while removing the index finger.



Courtesy of LMA North America, Inc.

FIGURE 5-9(G) Inflation without holding the tube allows the mask to seat itself optimally.

for complications such as regurgitation, laryngeal spasm, bronchospasm, coughing, retching, excessive salivation, and oxygen desaturation (Nunez et al., 1998; Samarkandi, 1998).

Limitations of LMA

✔ Positive pressure ventilation may be provided via most LMAs at peak inspiratory pressure of up to 20 cm H_2O (up to 30 cm H_2O with LMA-ProSeal).

Due to the unique position of its mask, rotation or turning of the LMA may cause misplacement of the mask and result in gastric insufflation and air leakage from the mask seal (Latorre et al., 1998). For this reason, the patient's head position and alignment of the LMA should be checked frequently to ensure adequate ventilation. Positive pressure ventilation may be provided via most LMA at peak inspiratory pressure of up to 20 cm H_2O (up to 30 cm H_2O with LMA-ProSeal). For patients with low compliance or high airflow resistance, the LMA may not be able to withstand the pressure, and air leaks may develop. When high peak inspiratory pressure is anticipated, the LMA should be replaced with an endotracheal tube (LMA North America, Inc., 1999, 2012).

The recommended time of LMA usage for each patient is 2 hours repetitive, with a maximum time from 6 to 8 hours in the hands of experienced users. For extended usage, a heat and moisture exchanger may be used to provide supplemental humidity to the airway. Since nitrous oxide diffusion through the silicone cuff of a reusable LMA can raise the cuff volume and pressure, the cuff pressure should be checked periodically and maintained at 60 cm H_2O. Excessive cuff pressure may lead to malposition of the LMA or other adverse outcomes (LMA North America, 2012). The LMA is not a secured airway and it does not protect the lower airway from aspiration. The esophagus, which lies posterior to the LMA, has complete access to the larynx, and thus regurgitation or aspiration is a potential complication (Norton, 1998).

✔ Most LMAs are disposable.

Most LMAs are disposable. The reusable version of the LMA is handmade, thus the cost is rather significant. With proper sterilization by steam **autoclave** and careful handling, it can be reused up to 40 times, as long as the mask is not damaged during use, handling, and steam autoclaving. Table 5-7 outlines the limitations of the LMA.

autoclave: A method of sterilization using steam pressure, usually at 250°F (121°C) for a specific length of time. (Up to 275°F or 135°C for reusable LMA's.

TABLE 5-7 Limitations of the Laryngeal Mask Airway

Rotation of the LMA may cause misplacement of mask, gastric inflation, and air leaks.
Cuff does not provide seal at airway pressures greater than 20 cm H_2O.
The LMA does not protect the lower airway from aspiration.
Reusable version is costly but it can be reused up to 40 times after cleaning and steam sterilization.
Steam autoclaving is the only recommended method of sterilization for reusable LMA.

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ESOPHAGEAL-TRACHEAL COMBITUBE (ETC)

esophageal-tracheal combitube (ETC): An airway that may be inserted into the esophagus or trachea.

✔ The ETC is inserted blindly either into the trachea or esophagus.

The **esophageal-tracheal combitube (ETC)**, also called pharyngealtracheal lumen airway and esophageal-tracheal airway, is a combination of esophageal and endotracheal tube in one unit (e.g., Combitube™, by Kendall-Sheridan Corporation, Argyle, NY). Due to its design, ventilation is possible when the ETC is inserted blindly either into the trachea or esophagus (Liao & Shalit, 1996). The ETC can be inserted easily by unskilled personnel (Yardy, Hancox & Strang, 1999), and it has been used successfully as an alternate artificial airway in patients outside the hospital (Blostein, Koestner & Hoak, 1998; Hoak & Koestner, 1997; Rumball & MacDonald, 1997). Ventilation is provided via a 15-mm airway connector at the proximal end of the ETC.

✔ Both lumens on the ETC can be used to provide ventilation. Lumen 1 is used when the tube enters the esophagus and lumen 2 is used when it is in the trachea.

There are two cuffs on the ETC, a proximal latex pharyngeal cuff (100 mL) and a PVC cuff (15 mL) near the distal end of the tube (Figure 5-10). Both lumens on the ETC can be used to provide ventilation. Lumen 1 is used when the tube enters the esophagus and the distal cuff seals off the esophagus. Lumen 2 is used when it is in the trachea and the proximal cuff seals off the trachea. Figure 5-11 shows the relative positions of the ETC when it enters the esophagus or trachea.

Insertion and Use of ETC

✔ Following blind intubation with a esophageal-tracheal combitube (ETC), ventilation should be attempted initially through lumen 1 of the ETC.

The ETC can be inserted with or without a laryngoscope. The tube is properly inserted once the black rings lie opposite the front teeth. After insertion, both cuffs are inflated immediately. Since the ETC is designed to provide ventilation when the tube is in the trachea or esophagus, it does not matter whether the tube enters the esophagus or trachea.

blind intubation: Insertion of an artificial airway without use of visual aid or under direct vision.

During **blind intubation**, the ETC is more likely to go into the esophagus. Therefore, ventilation through the ETC should be done initially via lumen 1. When the distal end of the ETC is in the esophagus, air goes through the side ports, becomes trapped between the cuffs, and is forced into the trachea. If ventilation via lumen 1

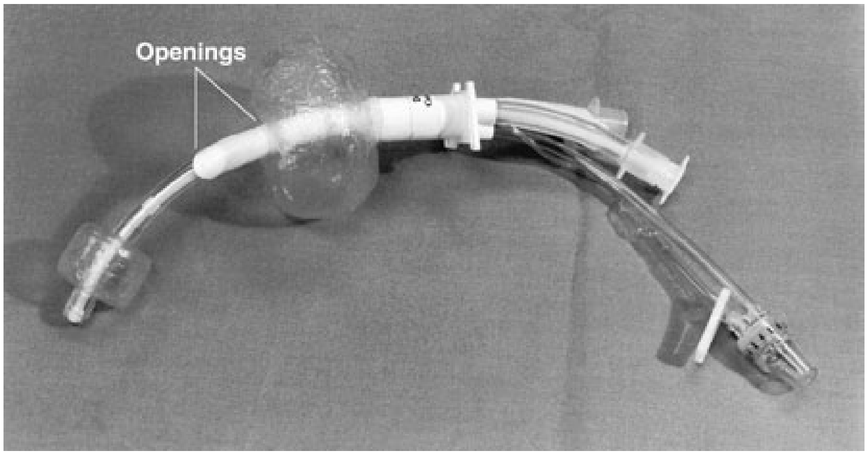

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FIGURE 5-10 A pharyngealtracheal lumen airway (PTLA).

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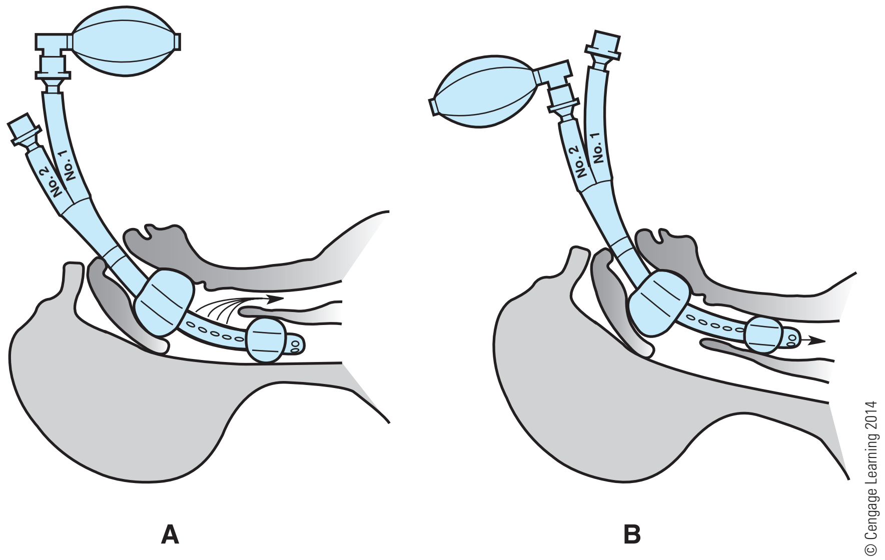

FIGURE 5-11 After placement of the pharyngealtracheal lumen airway, both cuffs are inflated. (A) When the tube enters the esophagus, lumen No. 1 is used to ventilate the patient via the openings between the cuffs. (B) When the tube enters the trachea, lumen No. 2 is used to provide ventilation directly into the trachea.

is absent or poor, lumen 2 should be used to provide ventilation, as the distal end of the ETC is likely in the trachea.

If ventilation is poor with lumens 1 and 2, a cuff leak may be present. This problem may be corrected by inflating the proximal cuff with more air. Try lumen 1 again and check for adequacy of ventilation. If ventilation is still poor, the entire procedure described earlier can be repeated after preoxygenating the patient.

Complications of ETC

Cases of complications associated with the use of the ETC have been reported. These complications are related to either hemodynamic stress or air leaks. In one report, the hemodynamic and catecholamine stress response after insertion of the ETC were significantly higher compared to laryngeal mask airway or endotracheal intubation. This observation might be attributed to the pressure of the pharyngeal cuff of the ETC (Oczenski et al., 1999). In another report, different types of air leak (subcutaneous emphysema, pneumomediastinum, and pneumoperitoneum) were observed as a result of using the ETC. Esophageal laceration appears to be the cause of these air leaks (Richards, 1998; Vézina et al., 1998).

DOUBLE-LUMEN ENDOBRONCHIAL TUBE (DLT)

double-lumen endobronchial tube: A special airway for independent lung ventilation. It has two separate lumens, two cuffs, and two pilot balloons.

The **double-lumen endobronchial tube (DLT)** has two separate lumens (tracheal and bronchial), two cuffs (tracheal and bronchial), and two pilot balloons (tracheal and bronchial). It is also known as the double-lumen tracheobronchial tube and may be either a left- or right-sided tube.

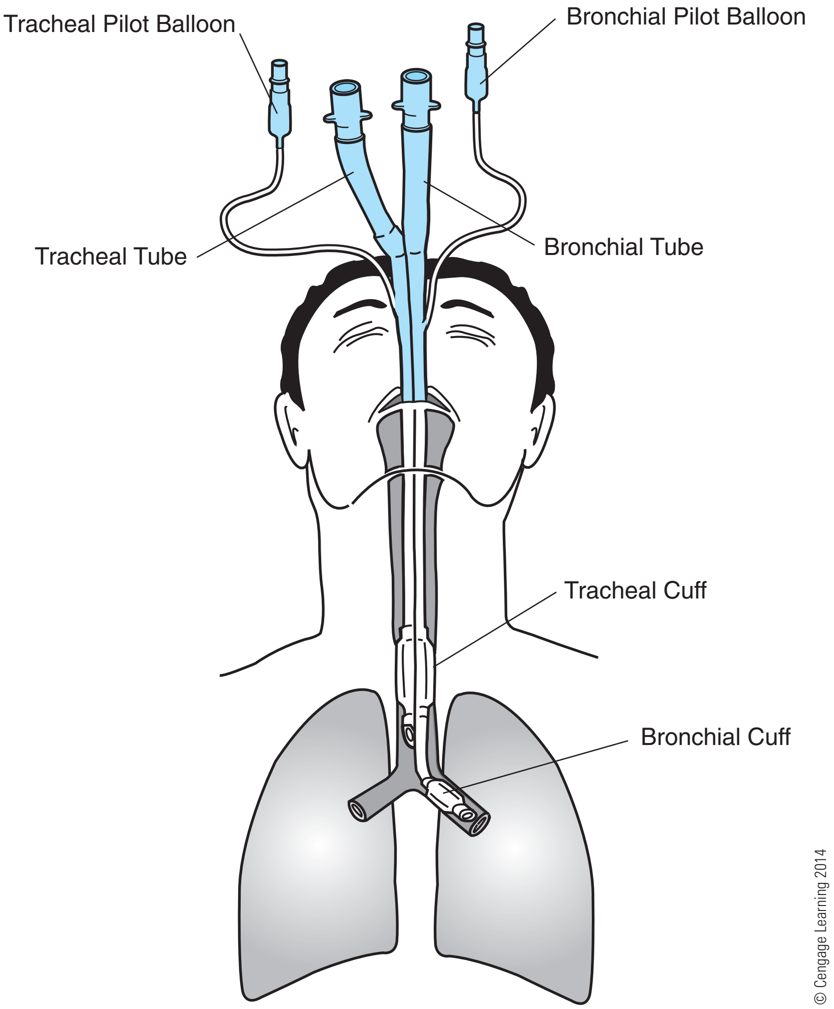

FIGURE 5-12 A left-sided double-lumen endobronchial tube (DLT) in working position with the tracheal and bronchial cuffs instead.

The left-sided tube is more commonly used than the right-sided tube. This is because precise placement of the right-sided tube is more difficult, as the right upper lobe bronchus is only about 2 cm from the carina of an adult. Should the distal end of the right-sided tube pass the right upper lobe bronchus, there would be no ventilation to the right upper lobe. Figure 5-12 shows a left-sided DLT in its working position with both cuffs inflated.

Indications

✔ Independent lung ventilation is done via a DLT to facilitate lung isolation, surgical procedure, and bronchopleural/bronchocutaneous fistulas.

There are three main uses of DLTs. They are used to provide independent lung ventilation where isolation of the lungs is desirable in order to prevent lung-to-lung spillage of blood or pus. They are also used to provide one-lung ventilation so that the nonventilated lung may undergo surgical procedure on the thoracic structures with minimal movement. In disease conditions such as bronchopleural

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and bronchocutaneous fistulas, DLTs can provide ventilation by overcoming the persistent air leak through the fistulas (Barash, 2001).

Selection of DLT

✓ It is essential to use the largest DLT appropriate for the patient.

It is essential to use the largest DLT appropriate for the patient since most complications associated with DLTs result from using small tubes. Recognizing the diameter of the bronchus (usually the left) is a crucial determinant of success in the use of a DLT. On a typical posterior-anterior (PA) chest radiography, the air bronchogram is enlarged by 10% as the bronchi are about 15 cm from the plate (Russell, 2004). If the bronchi are not visible on the chest radiography, the diameter of the left bronchus may be estimated by using 68% of the tracheal diameter. Table 5-8 shows the guidelines for choosing a left-sided DLT. In general, 28 and 32 French (Fr) DLTs are suitable for small children; 35 Fr for large children or small adults; and 37, 39, and 41 Fr for adults (Brodsky et al., 1997).

✓ Adult-sized DLTs range from 35 to 41.

Insertion of DLT

✓ The stylet should be removed as soon as the DLT has passed the vocal cords.

After the patient is anaesthetized and paralyzed, the DLT is inserted into the trachea using direct laryngoscopy until the tracheal cuff is just below the vocal cords. At this point, the DLT is about 6 cm from the final position. If a stylet is used to guide the DLT, it should be removed as soon as the DLT has passed the vocal cords. This maneuver minimizes the incidence of airway trauma caused by the stylet.

The bronchial cuff is then inflated in the trachea until the air leak stops on inflation. The bronchial adaptor is connected to the ventilator circuit and both lungs of the patients are ventilated via the bronchial tube.

TABLE 5-8 Guidelines for Choosing an Adult Left-Sided DLT

Measured Tracheal width (mm)	Predicted left Bronchus width (mm)	Double-Lumen Tube size	Outer Diameter (mm)	
			Main body	Left lumen
>18	>12.2	41 Fr	14–15	10.6
16.1–18	10.9–12.1	39 Fr	13–14	10.1
15.1–16	10.2–10.8	37 Fr	13–14	10.0
<15	9.5–10.1	35 Fr	12–13	9.5

Note: Tracheal width as measured from the PA chest radiography. Predicted left bronchus width is 68% of the measured tracheal width. (Brodsky et al., 1997).

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With each breath, the tube is advanced into the trachea until the bronchial segment plugs the bronchus. The endpoint signs are (1) resistance to advancement (even with low compliance lungs), (2) unilateral ventilation by observation and auscultation, and (3) reduction in compliance (e.g., ↑PIP in volume-controlled ventilation). At this point, the tube is about 2.5 to 3 cm (bronchial cuff length plus 1 cm) from its final position.

Once the bronchial plugging point has been identified, the bronchial cuff is deflated and the tube is inserted another 2.5 to 3 cm (bronchial cuff length plus 1 cm). The final bronchial cuff volume needed to seal should be small, about 1 to 2 mL. The patient connection is now changed to the tracheal connection. The tracheal cuff is inflated until a seal is made in the trachea. Proper positioning of the DLT can be verified by auscultation or fiberoptic bronchoscopy (Klein et al., 1998).

✓ Proper positioning of the DLT can be verified by auscultation or fiberoptic bronchoscopy.

If the left-sided tube goes right, turning the head 90° so that the chin is on the right shoulder and rotating the tube on its axis to restore the bronchial tube to point laterally to the left can usually achieve insertion into the left main bronchus (Russell, 2004).

Complications of DLT

✓ The most severe form of airway injuries caused by DLTs is airway rupture.

Use of DLTs may lead to airway injuries (Campos et al., 2000), and the most severe form of airway injuries is airway rupture. The incidence of airway rupture is higher when large and medium-sized red rubber DLTs are used. In contrast, small polyvinyl-chloride (PVC) DLTs have been associated with airway rupture (Akhtar, 1999). Some risk factors for airway rupture are listed in Table 5-9.

Table 5-9 Risk Factors for Airway Rupture

Risk Factor	Examples
Direct trauma	Forceful insertion Tube too large for the bronchus Tube advanced with stylet in place (questionable) Movement of tube while both cuffs are inflated Carinal hook damage
Cuff overinflation	Too rapid an inflation Too large a volume Nitrous oxide distention
Preexisting airway pathology	Congenital airway wall abnormalities Airway wall weakness from tumor infiltration or infection Airway distortion from mediastinal lymph nodes or tumor

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Some recommendations for the safe placement of a DLT (Fitzmaurice, 1999) include (1) choose the largest PVC DLT that is appropriate for the patient, (2) remove the bronchial stylet once the tip of the tube is past the vocal cords, (3) never overinflate either cuff and use 3-mL syringe to inflate the bronchial cuff, (4) when nitrous oxide is used, measure the cuff pressures intermittently and keep the bronchial pressure <30 cm H_2O, and (5) deflate the bronchial cuff when lung isolation is not required.

SUMMARY

Special airways discussed in this chapter are useful in situations where bag/mask ventilation is inadequate, endotracheal intubation is not readily achievable, or independent lung ventilation is required. A respiratory care practitioner should be knowledgeable and proficient in the use of these airways in different patient and clinical situations.

Self-Assessment Questions

1. During bag/mask ventilation of a 70-kg patient, the therapist encounters a great deal of airflow resistance during inspiration. The therapist should select an oropharyngeal airway with a length that is equal to the distance from the:

   A. center of the mouth to the angle of the jaw.
   B. corner of the mouth to the angle of the jaw.
   C. tip of the nose to the earlobe.
   D. ridge of the nose to the earlobe.

2. A nasopharyngeal airway may be used as an airway adjunct in all of the following conditions *except* patients with:

   A. intact gag reflex.
   B. oral trauma.
   C. trimus (lockjaw).
   D. epistaxis (nose bleed).

3. The normal size range of nasopharyngeal airway for adults ranges from:

   A. 6 to 8.
   B. 2 to 4.
   C. 4 to 6.
   D. 8 to 10.

4. An inward migration of a nasopharyngeal airway may be prevented by:

   A. using the larger size nasopharyngeal airway.
   B. using a safety pin on the distal end of the nasopharyngeal airway.
   C. leaving at least 2 inches of the nasopharyngeal airway outside the nare.
   D. using a nasal cannula to hold the nasopharyngeal airway in place.

5. Since an esophageal obturator airway (EOA) is inserted into the ________, the cuff at the distal end must be ________ before providing manual ventilation.

A. tracheal, deflated	C. esophagus, deflated
B. trachea, inflated	D. esophagus, inflated

6. Since there are two ports on the mask of the ________, the manual resuscitation bag must be attached to the ventilation port of this airway.

A. esophageal obturator airway	C. esophageal gastric tube airway
B. laryngeal mask airway	D. esophageal-tracheal combitube

7. The silicone-based laryngeal mask airway may be reused up to ________ times with proper handling and ________.

A. 40, chemical sterilization	C. 100, chemical sterilization
B. 40, steam autoclaving	D. 100, steam autoclaving

8. Since the cushioned mask of a laryngeal mask airway (LMA) provides a seal over the ________, it is not necessary for the LMA to enter the larynx or trachea.

A. tracheal opening	C. esophageal opening
B. laryngeal opening	D. vocal cords

9. Which of the following is *not* true concerning the laryngeal mask airway (LMA)?

A. LMA does not protect an airway from regurgitation and aspiration.
B. LMA is sterilized by steam autoclave.
C. Size 4 and size 5 LMAs are suitable for most adults.
D. LMA may withhold airway pressures up to 60 cm H_2O.

10. An esophageal-tracheal combitube (ETC) is inserted into the:

A. trachea.	C. trachea or esophagus.
B. esophagus.	D. esophagus or larynx.

11. Both lumens on the ETC can be used to provide ventilation. Lumen 1 is used when the tube enters the ________ and lumen 2 is used when it is in the ________.

A. trachea, esophagus	C. trachea, larynx
B. esophagus, trachea	D. esophagus, larynx

12. Following blind intubation with an ETC, ventilation should be attempted initially through lumen 1 of the ETC because:

A. an airway usually goes into the trachea during blind intubation.
B. an airway usually goes into the esophagus during blind intubation.
C. lumen 1 has a closed distal end.
D. lumen 2 has a closed distal end.

13. Clinical conditions that may require independent lung ventilation include all of the following *except*:
    - A. respiratory distress syndrome.
    - B. antibiotic-resistant left lung infection.
    - C. bronchopleural fistula.
    - D. right lung lobectomy.

14. Following proper insertion and placement of a double-lumen endobronchial tube:
    - A. only the bronchial cuff is inflated.
    - B. only the tracheal cuff is inflated.
    - C. both cuffs are inflated.
    - D. both cuffs are deflated.

Answers to Self-Assessment Questions

1. A.
2. D.
3. A
4. B.
5. D.
6. C.
7. B.
8. B.
9. D.
10. C.
11. B.
12. B.
13. A.
14. C.

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(A list of the articles used to develop the above guidelines is available by writing to the American Society of Anesthesiologists, 520 North Northwest Highway, Park Ridge, Illinois 60068–2573).

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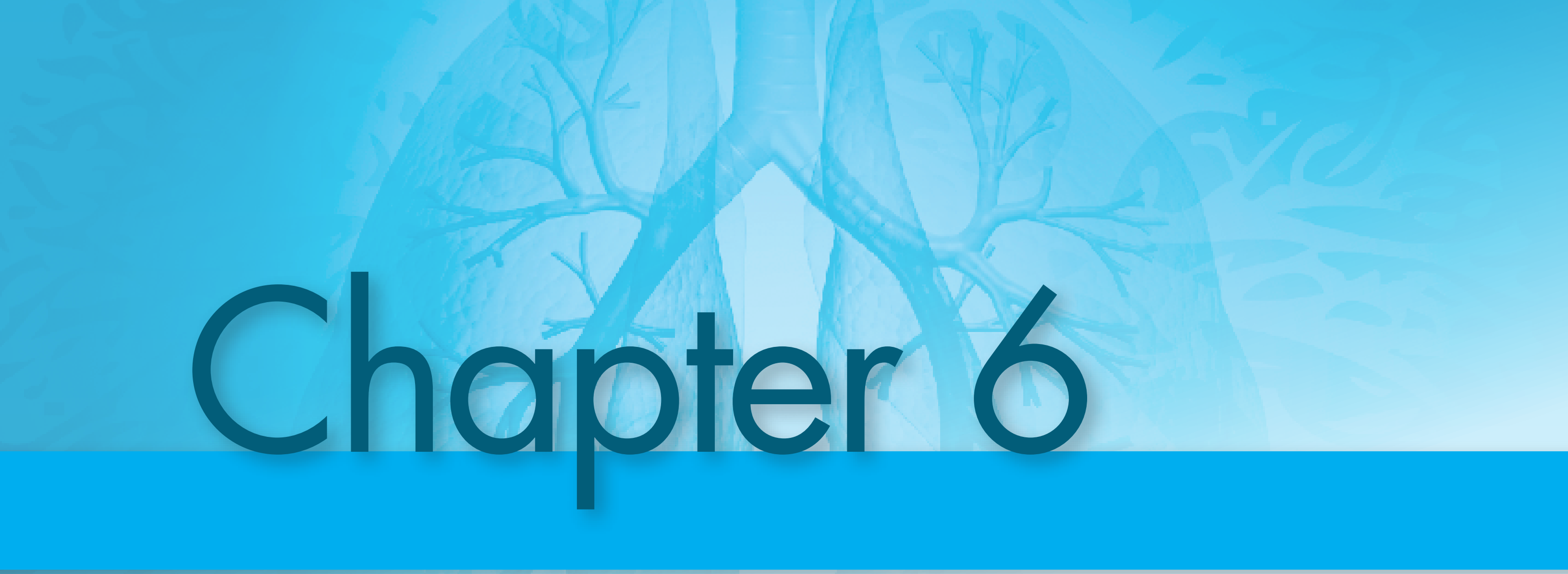

Chapter 6

Airway Management in Mechanical Ventilation

David W. Chang

Outline

- Introduction
- Intubation
  - *Indications*
- Common Artificial Airways in Mechanical Ventilation
  - *Endotracheal Tube*
  - *Tracheostomy Tube*
  - *Specialty Tracheostomy Devices*
- Intubation Procedure
  - *Preintubation Assessment and Signs of Difficult Airway*
  - *Supplies*
  - *Special Visualization Devices*
  - *Selection of Endotracheal Tube*
  - *Ventilation and Oxygenation*
  - *Oral Intubation*
  - *Nasal Intubation*
  - *Common Errors*
  - *Signs of Endotracheal Intubation*
  - *Signs of Esophageal Intubation*
- Rapid Sequence Intubation
  - *Indications and Contraindications*
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- Management of Endotracheal and Tracheostomy Tubes
  - *Securing Endotracheal and Tracheostomy Tubes*
  - *Cuff Pressure*
  - *Minimal Occlusion Volume and Minimal Leak Technique*
  - *Endotracheal Suctioning*
  - *Endotracheal Tube Changer*
- Speaking Valves
  - *Contraindications*
  - *Safety Requirements*
  - *Positive Pressure Ventilation*
- Extubation
  - *Predictors of Successful Extubation*
  - *Procedure*
  - *Unplanned Extubation*
- Complications of Endotracheal Airway
  - *During Intubation*
  - *While Intubated*
  - *Immediately after Extubation*
  - *Following Extubation*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

carina
endotracheal tubes
laryngoscope
Macintosh blade
Magill forceps
Mallampati classification
Miller blade
pilot balloon
radiopaque
rapid sequence intubation (RSI)
sniffing position
speaking valve
stylet
tracheostomy tube
unplanned extubation
vagus nerve
vallecula
vocal cords

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- List the indications for intubation.
- List the characteristics of an endotracheal tube and a tracheostomy tube.
- Describe the method to select an endotracheal tube and the procedure for oral and nasal intubation.
- Name the signs of endotracheal and esophageal intubation.
- Outline the methods to manage endotracheal and tracheostomy tubes.
- Describe the method to monitor and manage the cuff pressure.
- Describe the clinical use and safety requirements of speaking valves.
- Outline the predictors of successful extubation and the procedures for planned and unplanned extubation.

INTRODUCTION

In mechanical ventilation, artificial airways provide a vital link between the ventilator and the patient. Two common artificial airways used in conjunction with mechanical ventilators are the endotracheal (ET) and tracheostomy tubes. For these airways to work properly and efficiently, they must be used and maintained correctly. This chapter provides a practical presentation on oral and nasal intubation, suctioning, and extubation. Supplies commonly used for intubation and suctioning are also included.

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INTUBATION

✓ The surgical procedure that creates an opening at the trachea is called *tracheotomy*.

tracheostomy tube: An artificial airway inside the trachea that is inserted through a surgical opening at the trachea.

Endotracheal (ET) intubation is the placement of an ET tube inside the trachea through the mouth or nostril. It is estimated that 15 million patients undergo ET intubation annually (Coppolo et al., 1990).

ET intubation is a simpler procedure than tracheotomy—a surgical procedure that creates an airway opening by cutting into the trachea. Compared to an ET tube (Figure 6-1), a **tracheostomy tube** (Figure 6-2) is much shorter and it

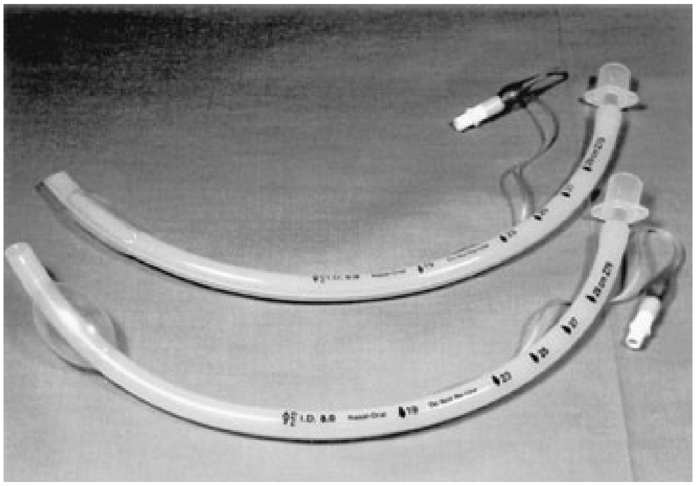
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FIGURE 6-1 Two adult endotracheal tubes (8.0 mm ID). Note that one's cuff is inflated and the other's is not. Also note the markings visible on the tubes.

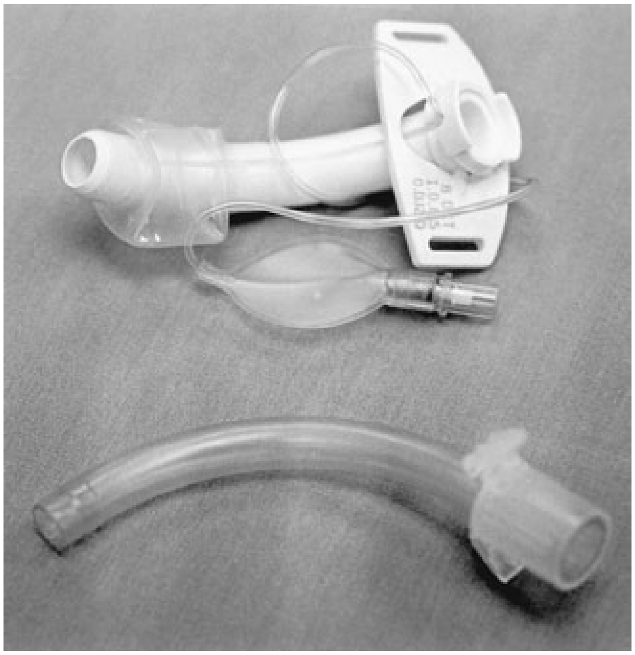
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FIGURE 6-2 An adult tracheostomy tube with a disposable inner cannula that may be replaced as required to maintain patency.

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provides closer access to the lower airways. It has a lower mechanical deadspace volume than an ET tube. It also ventilates the patient more efficiently and enhances secretion removal. In spite of many advantages of a tracheostomy tube, ET intubation is preferred as the initial means of establishing an artificial airway.

Oral and nasal intubations are commonly done by respiratory care practitioners. When these two routes are not accessible or when the need for a long-term artificial airway is expected, a tracheotomy is done by a physician who is proficient in this surgical procedure.

Indications

The decision to perform ET intubation versus tracheotomy is based on the expected duration of need. In general, if the patient requires an artificial airway for a brief period (e.g., 10 days or less) and full recovery is expected, an ET tube is used. On the other hand, if the patient's condition is critical and recovery is not expected any time soon (e.g., more than 21 days), a tracheostomy tube is preferred (Shapiro et al., 1991).

Choosing when to intubate is also a difficult clinical decision because delayed intubation may lead to hypoventilation, hypoxemia, and hypoxia. The timing of intubation can be based on four indications: (1) relief of airway obstruction, (2) protection of the airway, (3) facilitation of suctioning, and (4) support of ventilation (Shapiro et al., 1991). Some examples for each of these indications are listed in Table 6-1.

TABLE 6-1 Indications for Using Artificial Airway

Indication	Examples
Relief of airway obstruction	Epiglottitis Facial burns and smoke inhalation Vocal cord edema
Protection of the airway	Prevention of aspiration Absence of coordinated swallow
Facilitation of suctioning	Excessive secretions Inadequate cough
Support of ventilation	Ventilatory failure / respiratory arrest Chest trauma Postanesthesia recovery Hyperventilation to ↓ intracranial pressure

(Data from Shapiro et al., 1991; White, 2002; Whitten, 1997.)

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COMMON ARTIFICIAL AIRWAYS IN MECHANICAL VENTILATION

✓ Oral intubation is easy to perform and is often done in emergency situations.

An ET tube may be inserted orally (oral intubation) or nasally (nasal intubation) through the larynx into the trachea. Oral intubation is easy to perform and it is often done in emergency situations. Nasal intubation is more time-consuming and it is more suitable in elective intubations.

Some ET tubes (e.g., Spiral-Flex®) are reinforced with spiral stainless steel wire within the tube wall to reduce risk of tube kinking. They are the ideal choice for head and neck surgery when bending or compression of the tube is likely to occur, or for patients in the ICU biting on the tube or experiencing seizures. These tubes are part metal and must be replaced with a regular ET tube when the patient undergoes any procedure involving magnetic resonance imaging.

Endotracheal Tube

✓ An oral route of intubation allows the passage of a larger ET tube than nasal intubation.

✓ A larger ET tube lowers the peak, plateau, and mean airway pressures.

Intubation through the mouth is the preferred method of establishing an artificial airway (Figure 6-3). An oral route provides quick access to the lungs in emergency situations and it allows the passage of a larger ET tube than the nasal route. A larger tube has less airflow resistance, and it lowers the airway pressure requirements. However, oral intubation is less comfortable to the patient and may cause gagging and excessive secretion production. Agitated patients may bite down on the tube and cause airflow obstruction. In general, the oral route of intubation may be preferred for cardiopulmonary resuscitation, apneic patient, nasal trauma, nasopharyngeal obstruction, midfacial trauma, basilar skull fracture, epiglottitis, and anticipation of fiberoptic bronchoscopy.

Another common ET tube (e.g., Hi-Lo Evac) has an evacuation lumen/port that allows continuous aspiration of subglottic secretions. Studies have shown that

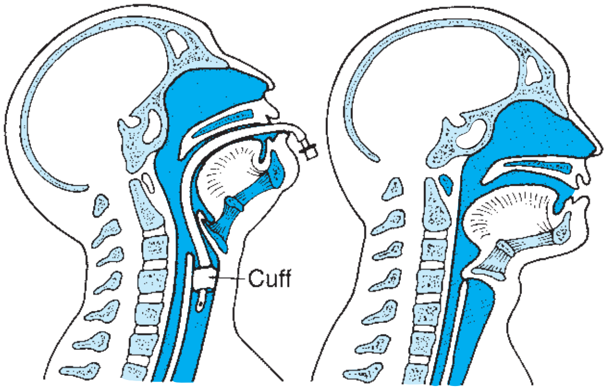

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FIGURE 6-3 This illustration shows how an inflated cuff seals the trachea.

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use of this type of tube delays the early onset of ventilator-associated pneumonia (Valle et al., 1995) and prevents nosocomial pneumonia in intubated patients (Mahul et al., 1992).

Intubation through the nostril is better tolerated by the patient. It provides an ideal access to the lower airway in conditions where oral access is limited. The nasal route of intubation may be preferred for trismus (lockjaw), oral trauma or deformities, mandible fracture, and short neck.

✓ Disadvantages of nasal intubation include difficulty to insert, use of a smaller ET tube, and potential development of sinusitis.

The disadvantages of nasal intubation include difficulty to insert, use of a smaller ET tube, and potential development of sinusitis.

Tracheostomy Tube

✓ The decision to change from an ET tube to a tracheostomy tube is based on the patient's condition and prognosis.

A tracheostomy tube is inserted through a surgical opening into the trachea. In long-term mechanical ventilation, it is used to replace the ET tube that has been in place for 21 days or longer (Shapiro et al., 1991).

The tracheostomy tube bypasses the upper airway and the glottis and therefore avoids any potential injury in these areas and offers lower airflow resistance. It is also easier to maintain, stabilize, and suction. In addition, the patient can eat and drink with the tracheostomy cuff properly inflated (Shapiro et al., 1991).

✓ Sterile technique must be followed during tracheostomy tube care and suctioning.

Tracheostomy tubes are not without drawbacks. Since a tracheostomy tube is inserted through a surgical opening, infection and trauma to the surgical site are always a threat. To reduce the potential complications of tracheostomy, sterile and aseptic techniques must be followed during tracheostomy care and suctioning (White, 2002).

Specialty Tracheostomy Devices

There are two main types of tracheostomy tubes: cuffed and cuffless. Both cuffed and cuffless tubes are available with or without inner cannulas. Disposable tracheosotmy tubes are made of PVC plastic or silicone. Reusable tubes are made of silver or stainless steel. Specialty tracheostomy tubes are also available to suit different patient requirements. Some examples are discussed below.

Talking tracheostomy tube. Trach-Talk™ Tracheostomy Tubes (Portex, 2011) was designed to assist the patient to speak in a low whispered voice. With the cuff inflated, a gas line with a thumb port is connected to a gas source (air or oxygen). The flow of gas is set from 4 L/min to 6 L/min. When the thumb port is occluded, the gas passes through the vocal cords making phonation possible.

Speaking valves are adaptors used on a regular tracheostomy tube that make phonation possible without the need of a constant gas flow. Speaking valves will be discussed later in this chapter.

Tracheostomy tube with high volume-low pressure cuff. The high volume-low pressure cuff (e.g., Bivona Fome-Cuf®) uses a silicone foam material to fill the cuff. This type of foam cuff does not require manual inflation with a syringe. Rather, the self-inflating nature of the foam rubber provides a continuous seal while maintaining minimal tracheal wall pressure.

The cuff of the Bivona Fome-Cuf® tube can be inflated either by attaching the red wing pilot port to the side port auto control airway connector (if available) or by leaving the red wing pilot port open to room air for self-inflation. The important point is to check for cuff leak or obstruction. Cuff leak is evident when the gas leak is audible and the expired tidal volume is lower than the set tidal volume. Cuff obstruction may be present when the airway pressures are higher than the baseline measurement. In both cases, the patient's vital signs and oxygen saturation would show corresponding changes.

✓ The tracheostomy button is used to maintain the stoma of a patient on a temporary or permanent basis.

Tracheostomy button. The tracheostomy button is used to maintain the stoma of a patient on a temporary or permanent basis. The button offers several advantages. Direct access to the trachea facilitates tracheal suctioning and removal of secretions. In emergency situations, the button can be replaced with a traditional tracheostomy tube without the need for another tracheotomy. The buttons are also suitable for patients who may require repeated tracheostomies (e.g., myasthenia gravis, quadriplegia).

INTUBATION PROCEDURE

Intubation is a fairly simple procedure. In order to become proficient in this procedure one may need to exercise good organization and frequent practice. The procedure described below provides the basics and it may vary somewhat depending on the preference of an individual and existing protocol of the respiratory therapy department.

Preintubation Assessment and Signs of Difficult Airway

Prior to intubation, the patient must be assessed to rule out any potential contraindications to include head injury, cervical spine injury, airway burns, and facial trauma (Finucane et al., 2010). Anesthesia consultation is advised in cases of unfamiliarity or difficult intubation.

Mallampati classification: A method to evaluate the degree of difficulty in intubation.

The degree of difficulty in intubation due to anatomical structures can be evaluated by using the **Mallampati classification** method (Figure 6-4). This method is based on the anatomical structures visible with the mouth wide open and tongue protruded in a sitting position. Ease of oral intubation ranges from Class 1 (easiest) to Class 4 (most difficult) (Table 6-2) (Finucane et al., 2010).

Other signs of difficult airway include: increased size of tongue in proportion to pharyngeal size, neck mass, anterior larynx position, decreased mandibular

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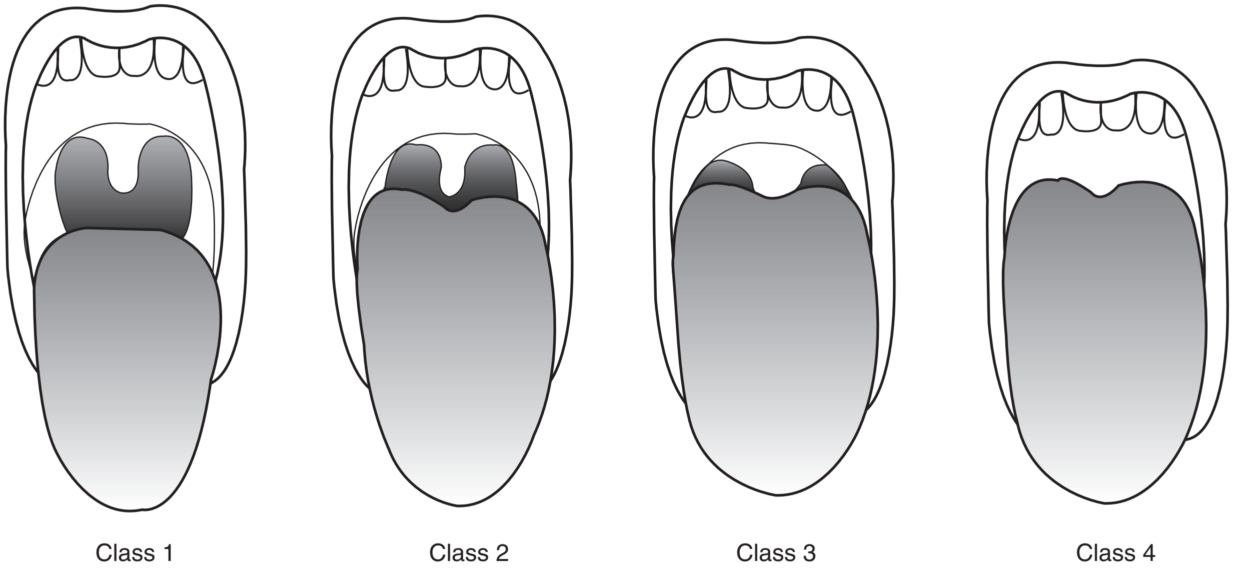

FIGURE 6-4 Mallampati classification.

laryngoscope: An instrument that is used to displace the tongue and soft tissues, and visualize the larynx and vocal cords during endotracheal intubation.

stylet: A flexible but semirigid wire placed inside an endotracheal tube to provide it with a desired curvature.

Magill forceps: Special forceps used to perform nasal intubation under direct vision.

space, reduced mouth opening, limited head extension, and dental overbite (Rich, 2005).

Supplies

The minimum supplies needed for ET intubation include (1) **laryngoscope** handle, (2) blade, (3) ET tube, (4) 10-mL syringe, (5) water-soluble lubricant, (6) tape, and (7) stethoscope. Optional supplies for ET intubation include (8) **stylet**, (9) topical anesthetic, and (10) **Magill forceps**.

TABLE 6-2 Mallampati Classification and Interventions

Mallampati	Able to See	Intervention
Class 1	Soft palate, fauces, uvula, anterior, and posterior tonsillar pillars	Conscious sedation
Class 2	Soft palate, fauces, and uvula	Conscious sedation
Class 3	Soft palate and base of uvula	Seek anesthesia consultation
Class 4	Soft palate only	Seek anesthesia consultation

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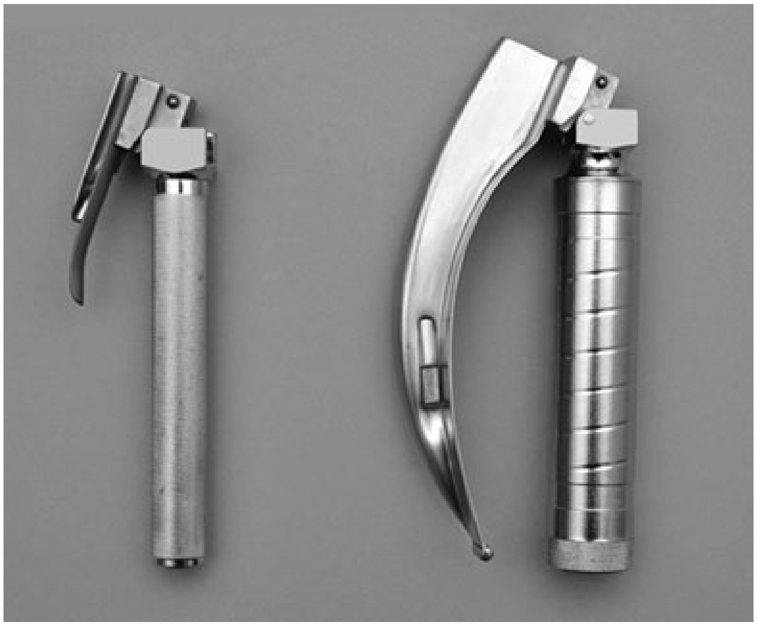

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FIGURE 6-5 A conventional laryngoscope with a Miller blade (left) and a Macintosh blade (right).

Miller blade: A straight laryngoscope blade. It lifts up the epiglottis for visualization of the vocal cords during endotracheal intubation.

MacIntosh blade: a curved laryngoscope blade. Its tip is placed at the vallecula and indirectly lifts the epiglottis for visualization of the vocal cords.

✓ The laryngoscope handle is held by the left hand.

✓ Laryngoscope blades range from size 00 for small preemie to size 4 for large adults.

✓ A straight blade lifts the tongue *and* epiglottis upward to expose the vocal cord and related structures.

✓ A curved blade lifts the tongue only.

vallecula: An area between the base of the tongue and epiglottis; an anatomical landmark for the placement of the curved blade.

In addition to the intubation supplies, proper airway management also requires oral airway, nasal airway, oxygen supply, and resuscitation bag/mask system.

Laryngoscope handle. The **laryngoscope** handle contains batteries and it allows attachment and manipulation of the blade during intubation. Figure 6-5 shows a laryngoscope handle with a **Miller blade** and a **Macintosh blade**.

The laryngoscope handle is held in the left hand since all standard blades attached to the handle are designed for right-hand intubations.

Blade. The laryngoscope blade attaches its flange onto the post of the handle (Figure 6-6). Once snapped into position, the built-in light source at the distal end of the blade comes on. A laryngoscope blade is either straight or curved and ranges from size 00 (small preemies) to 4 (large adults). Size 3 blades are intended for most adults. The straight blade (Miller blade) is used to lift up the epiglottis during intubation. The curved blade (Macintosh blade) is placed in an area called **vallecula,** and indirectly lifts the epiglottis for visualization of the vocal cords.

The basic technique of intubation is the same no matter which type of blade is used. The primary difference between these two blades is that a straight blade lifts the tongue and epiglottis upward to expose the vocal cords and related structures (Figure 6-7). The epiglottis is not visible when a straight blade is used correctly. The tip of a curved blade rests at the vallecula (between base of tongue and epiglottis) and lifts the tongue only (Figure 6-8). The epiglottis may be seen through the mouth when a curved blade is used correctly.

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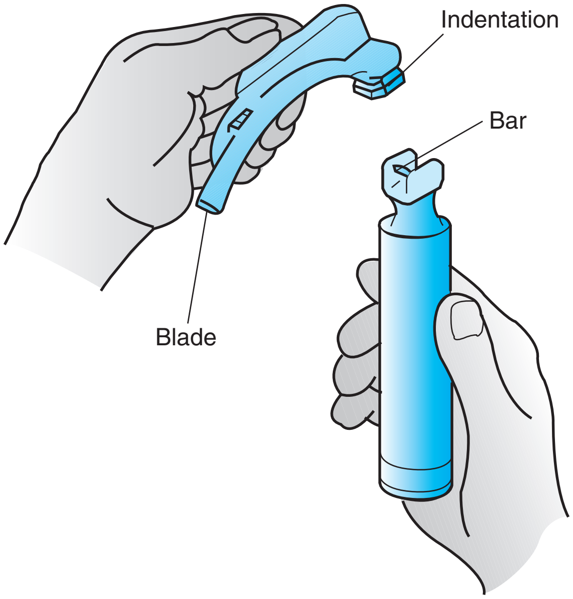

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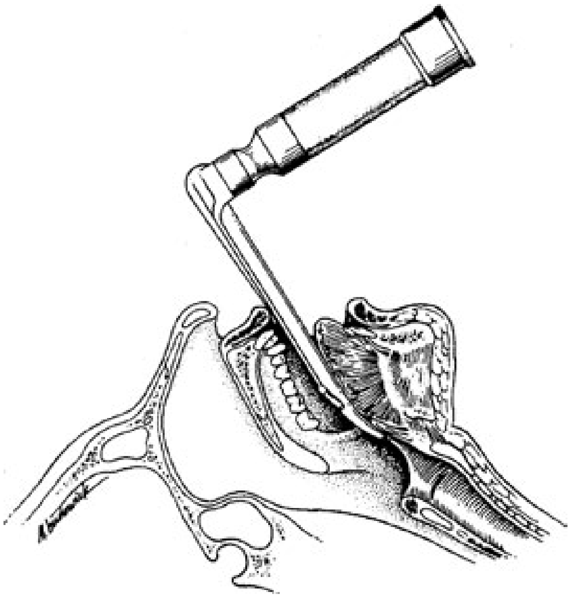
FIGURE 6-6 Attaching the laryngoscope blade to the handle. The blade locks into place when it is properly engaged.

✓ A straight blade functions better in patients with short necks, high or rigid larynxes, or obesity.

Patients with short necks, high larynxes, or who are obese often need straight blades to displace the tongue and attached soft tissues upward. A straight blade also functions better in patients with rigid larynxes due to scar formation or trauma (Whitten, 1997).



From Finucane, B. T., & Santora, A. H. (2003), Principles of airway management (3rd ed.). New York: Springer-Verlag. Used with permission.

FIGURE 6-7 Proper placement of a straight (Miller) blade during intubation. The blade lifts up the epiglottis anteriorly to expose the glottic opening and vocal cords.

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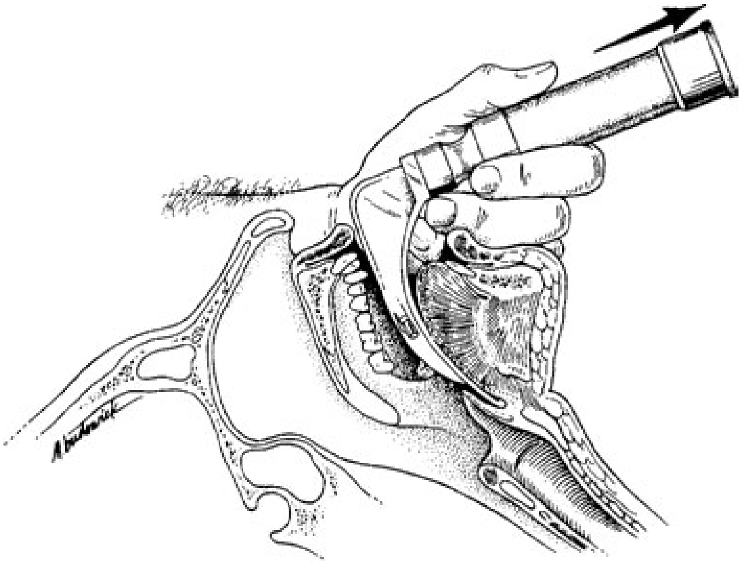

FIGURE 6-8 Proper placement of a curved (McIntosh) blade during intubation. The tip of blade is in the vallecula (between base of tongue and epiglottis). The epiglottis is elevated by using a forward-upward lift to expose the glottic opening and vocal cords.

From Finucane, B. T., & Santora, A. H. (2003). Principles of airway management (3rd ed.). New York: Springer-Verlag. Used with permission.

In general, a size 7.5 or 8.0 ET tube should be used for oral intubation of an adult male. For an adult female, a 7.0 or 7.5 should be used.

When an ET tube is used for nasal intubation, the tubes should be 0.5 mm to 1.0 mm *smaller* than the size selected for the oral route.

A curved blade is easy to learn to use as it can be easily positioned by advancing to the base of the tongue. Once the tongue is lifted upward, the epiglottis moves upward with the attached soft tissues thus exposing the **vocal cords.**

Despite one's training and preference, it is essential to gain experience and proficiency in using both types of laryngoscope blades, since in some emergency situations, the preferred type of blade may not be readily available.

Endotracheal tube. **Endotracheal tubes** come in sizes ranging from 2 to 10. The size refers to the internal diameter (ID) of the tube in millimeters (mm) and it comes in 0.5-mm increments. To reduce airflow resistance, the largest size appropriate to a patient should be used. In general, a size 7.5 or 8.0 ET tube should be used for oral intubation of an adult male. For an adult female, a 7.0 or 7.5 should be used. When an ET tube is used for nasal intubation, the tubes should be 0.5 mm to 1.0 mm *smaller* than the size selected for the oral route.

The proximal end of an ET tube has a 15-mm adaptor that fits all standard ventilator circuits and aerosol therapy adaptors. Along the body of the tube, a **radiopaque** line runs lengthwise for the verification of tube location by chest radiograph. Markings in centimeters (cm) are also shown along the tube for easy determination of the depth of intubation. The volume of air in the cuff at the distal end of the ET tube is controlled by using a large (10-mL or larger) syringe via the **pilot balloon.**

The ET tube is normally held in the right hand with the curvature facing forward. When intubating a spontaneously breathing patient, the tube is advanced into the trachea during spontaneous inspiratory efforts (when the vocal cords are opened

vocal cords: Two thin, almost parallel folds of tissue within the larynx that vibrate as air passes between them; an important landmark as the entry point to the trachea during intubation.

endotracheal tubes: An artificial airway inside the trachea that is inserted through the mouth or nostril.

radiopaque: Impenetrable to X-rays. It appears as a light area on the radiograph.

pilot balloon: The small balloon on the proximal end of an endotracheal or tracheostomy tube. It is used to regulate the volume of air in the cuff and to serve as an indicator of air volume in the cuff.

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wide). During expiration, the ET tube may bounce off the closed vocal cords and enter the esophagus.

For adult ET tubes, the syringe used to inflate the cuff should have a capacity of 10 mL or larger.

10-mL syringe. A syringe with a capacity of 10 mL or larger is used to test the pilot balloon and ET tube cuff before intubation and to inflate the cuff after intubation. After testing the integrity of the pilot balloon and cuff, air is withdrawn from the cuff to the syringe. The air-filled syringe may be left attached to the pilot balloon for rapid inflation of the cuff immediately after intubation.

Use only a water-soluble lubricant on the distal end of an ET tube.

Water-soluble lubricant. A water-soluble lubricant is used to lubricate the distal end of the ET tube for easy insertion into the trachea. Petroleum or oil-based lubricants must not be used in ET intubation. Once entering the lungs, they can cause adverse reactions to the airways and lung parenchyma.

Petroleum or oil-based lubricants must not be used as they can cause adverse reactions to the lungs.

Tape. Tape is used to secure the ET tube so that the tube will not move too high causing inadvertent extubation, or too low leading to main-stem intubation. Benzoin or other commercially available solutions may be effective in making the tape more adhesive to the damp skin. Zinc oxide base tape (by Hy Tape Corporation, New York) also sticks well to the skin when it is exposed to moisture.

If the ET tube is not secured properly, inadvertent extubation or main-stem intubation may result.

Stethoscope. A stethoscope is needed to auscultate bilateral breath sounds immediately after intubation.

Stylet. A flexible stylet wire guide is placed inside the ET tube to form a desired curvature and to make it more rigid for ease of intubation. Use of a stylet is not required for successful oral intubation. A stylet is not used in nasal intubation.

When a stylet is used, make certain that its end does not extend below the tip of the ET tube because the stylet can traumatize the tracheal wall. As a standard practice, the portion of stylet extending from the proximal end of the ET tube (outside the patient's mouth) is bent before intubation to prevent it from slipping deep inside the ET tube.

A stylet is not required for successful oral intubation and it is not used in nasal intubation.

Topical anesthetic. A topical anesthetic with decongestant (e.g., such as 4% lidocaine with oxymetazoline) may be used to numb and vasoconstrict the mucosal membrane. Use of a topical anesthetic is not feasible in emergency intubation or necessary in unconscious patients. It is useful to reduce the incidence of bronchospasm and vomiting when elective intubation is done in conscious and alert patients.

Magill forceps are used to perform nasal intubation.

Magill forceps. Magill forceps are used to perform nasal intubation. After the ET tube has been inserted through the nostril and becomes visible through the mouth, the laryngoscope blade and Magill forceps are used together to guide the ET tube into the trachea under direct vision.

Special Visualization Devices

A fiberoptic endoscope was used to perform nasal intubation as early as 1967 (Murphy, 1967). The first noncommercial fiberoptic laryngoscope was introduced in 1974 by American Optical. Currently, there are different types of visualization devices for intubation, including the low-cost lighted stylet, flexible fiberoptic stylets, and video systems (Liem et al., 2003). When compared to direct laryngoscopy, the optical stylet has equivalent intubation time, less hemodynamic variability, less trauma and sore throat, and less failed intubation of difficulty airway. The optical stylet does not require new skills and is easy to learn. It also offers a faster setup than the traditional fiberoptic bronchoscope and a low acquisition and maintenance cost. (Gravenstein et al., 2004).

Selection of Endotracheal Tube

Mentally count from 1 to 30 when you begin the intubation attempt.

Intubation attempts lasting longer than 30 sec may cause hypoxia and arrhythmias.

The size of an ET tube should be the largest one appropriate to a patient. Compared to a smaller ET tube, a larger one offers lower airflow resistance, and lower (peak, plateau, mean) airway pressures. A larger ET tube also improves dynamic compliance and facilitates secretion removal.

Table 6-3 shows the estimated size of the ET tube based on body size. In addition to the body weight or body size, final selection of an ET tube should be based on the clinical condition and tolerance of the patient.

Ventilation and Oxygenation

Before each intubation attempt, the patient must be adequately ventilated and oxygenated. If the patient is not breathing spontaneously, a resuscitation bag/mask system is used to provide ventilation and oxygenation (Figure 6-9).

If an intubation attempt is not successful after 30 sec, the ET tube and laryngoscope blade should be removed immediately and the patient ventilated with a bag/mask system and 100% oxygen. Ventilation and oxygenation should continue for at

TABLE 6-3 Estimation of ET Tube Size

Patient	Estimated Size*
Neonate (< 1000 grams)	2.5 mm ID
Neonate (1000 to 2000 grams)	3.0 mm ID
Neonate (2000 to 3000 grams)	3.5 mm ID
Neonate (> 3000 grams)	4.0 mm ID
Child (1 to 2 years)	4.5 mm ID
Child (2 to 12 years)	4.5 + (age/4) mm ID
Adult female	7.0 or 7.5 mm ID
Adult male	7.5 or 8.0 mm ID

**<size 6 uncuffed, size 6 cuffed or uncuffed, size 6 cuffed (Graber, 2004).*

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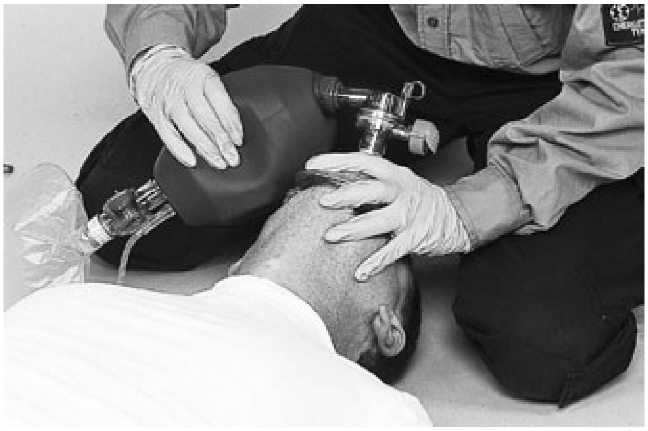
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FIGURE 6-9 Correct use of a mask to manually ventilate a patient.

least 30 sec or until the pulse oximetry (SpO_2) reading returns to a satisfactory level (e.g., $SpO_2 > 95\%$).

Oral Intubation

✔ The proper depth of an oral ET tube is guided by the distance marking (e.g., 22 cm) on the ET tube at the lips or incisors.

The sequence outlined in Table 6-4 provides a general procedure for oral intubation. The procedure should be modified to suit individual situations and to comply with existing protocols. The proper depth of an oral ET tube is guided by the distance marking (e.g., 22 cm) on the ET tube at the lips or incisors.

Nasal Intubation

The procedure for nasal intubation is similar to that for oral intubation. In nasal intubation, the ET tube is inserted through the nostril and then guided by the Magill forceps into the trachea (Table 6-5).

✔ "Blind" nasal intubation is done by advancing the ET tube slowly during spontaneous inspiratory efforts by listening for air movement through the ET tube.

✔ The proper depth of a nasal ET tube is guided by the distance marking (e.g., 26 cm for adult females and 28 cm for adult males) on the ET tube at the lips or incisors.

Blind Intubation. In alert and cooperative patients who are breathing spontaneously, "blind" nasal intubation may be done by inserting the ET tube into a nostril and advancing it slowly during inspiratory efforts. When the distal end of the ET tube approaches the trachea, air movement can be heard through the ET tube. The proper depth of a nasal ET tube is guided by the distance marking (e.g., 26 cm for adult females and 28 cm for adult males) on the ET tube at the lips or incisors (Reed et al., 1997). Breath sounds and a chest radiograph are done to confirm proper depth of the ET tube.

Common Errors

Errors can occur when intubation is done in a hurried fashion. They are also more likely to occur when it is done by someone who is not proficient or experienced with the intubation procedure. By staying calm during an intubation attempt and updating the intubation skills in a controlled setting (e.g., in operating room), errors can be minimized or avoided.

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TABLE 6-4 Procedure for Oral Intubation

1. Assemble and test supplies (e.g., check light source and ET tube cuff for air leak).
2. Lubricate the deflated cuff with a water-soluble lubricant.
3. Inform or explain procedure to patient.
4. Bag-mask ventilate and preoxygenate patient with 100% oxygen.
5. Tilt the head back and place in the **sniffing position** (Figure 6-10).
6. Open mouth, apply anesthetic spray.
7. Hold laryngoscope handle with left hand and insert blade into the right side of the opened mouth.
8. Slide blade to the base of tongue and sweep blade to the left.
9. Maneuver the tip of straight blade underneath the epiglottis (or the tip of curved blade at the vallecula).
10. Lift handle and blade up anteriorly to displace the tongue and attached soft tissues (Figure 6-11).
11. Locate the epiglottis (only with curve blade), larynx, and vocal cords (Figure 6-12).
12. Insert ET tube through the vocal cords under direct vision.
13. For adults, the centimeter marking on the ET tube should initially be placed at the lips or incisors at 21 to 23 cm.
14. Inflate cuff and confirm endotracheal tube placement (e.g., loss of phonation, rising SpO_2, presence of bilateral breath sounds and expired CO_2).
15. Verify proper depth of ET tube placement (1.5 inch above carina) with chest radiograph.

sniffing position: An ideal head position for endotracheal intubation. It is done by tilting the forehead back slightly and moving the mandible anteriorly to the patient.

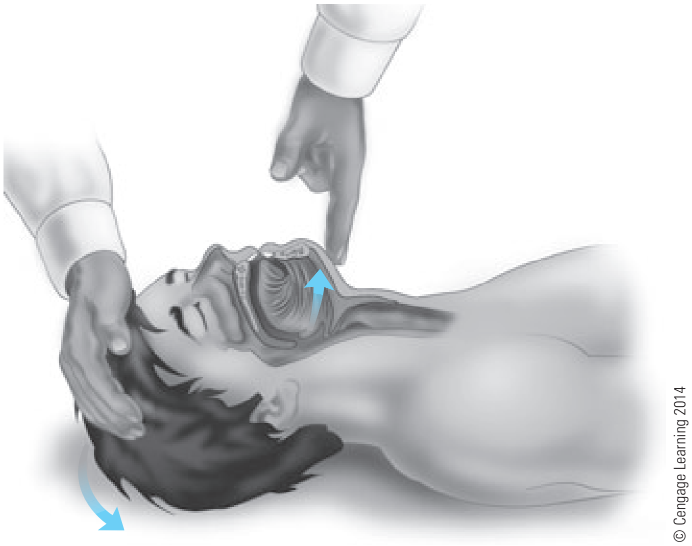
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FIGURE 6-10 The head tilt (sniffing) position is done by tilting the forehead back slightly and moving the mandible anteriorly to the patient.

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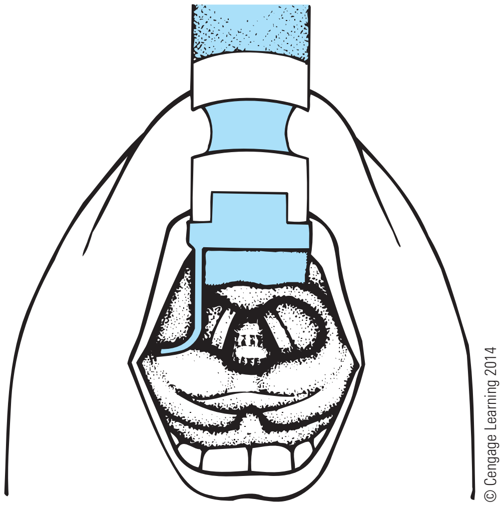
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FIGURE 6-11 Anatomy of the upper airway as viewed with a laryngoscope in place.

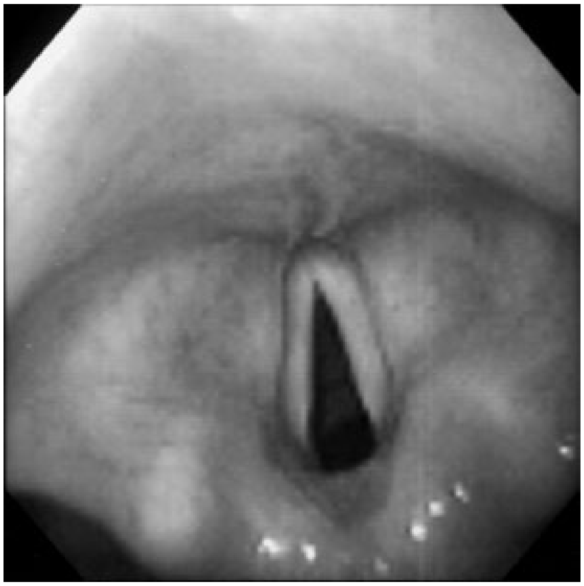
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FIGURE 6-12 Anatomical structures seen during direct laryngoscopy.

Table 6-6 outlines some problems that may be encountered during an intubation attempt. The potential cause and solution to each problem are provided.

Signs of Endotracheal Intubation

✔ Rising SpO_2, presence of bilateral breath sounds, airflow, condensations on ET tube, and detection of CO_2 are signs of successful ET intubation.

After intubation and inflation of the cuff, correct placement of the ET tube in the trachea must be checked immediately. If the patient is breathing spontaneously, bilateral breath sounds should be heard. Speech will not be possible since the vocal cords are bypassed by the ET tube and no longer receive airflow for making sound. In addition, airflow may be felt over the ET tube opening. With adequate ventilation

TABLE 6-5 Procedure for Nasal Intubation

1. Assemble and test supplies (e.g., check light source and ET tube cuff for air leak).
2. Lubricate the deflated cuff with a water-soluble lubricant.
3. Inform or explain procedure to patient.
4. Bag-mask ventilate and preoxygenate patient with 100% oxygen.
5. Tilt the head back and place in the sniffing position.
6. Spray anesthetic into nares (mixture of oxymetazoline and 4% topical lidocaine; up to 5 mL of lidocaine).
7. Insert 32 French soft nasal trumpet prelubricated with 2% lidocaine jelly.
8. Spray anesthetic through trumpet and remove trumpet.
9. Insert ET tube into a nostril and advance slowly until the distal end is near the tongue.
10. Open mouth and insert blade into the right side of mouth.
11. Slide blade to the base of tongue and sweep blade to the left.
12. Lift handle and blade up anteriorly to displace the tongue and attached soft tissues.
13. Locate the epiglottis, larynx, and vocal cords.
14. Use right hand to insert Magill forceps into mouth and guide ET tube through the vocal cord under direct vision.
15. The centimeter marking on the ET tube should initially be 26 cm at nare for women and 28 cm for men.
16. Inflate cuff and confirm endotracheal placement (e.g., loss of phonation, rising SpO_2, presence of bilateral breath sounds and expired CO_2).
17. Verify proper depth of ET tube placement (1.5 inch above carina) with chest radiograph.

and perfusion, the pulse oximetry measurements should show immediate and often drastic improvement. Moisture or condensation will form inside the ET tube on exhalation. A carbon dioxide (CO_2) indicator or end-tidal CO_2 monitor may be attached to the end of the ET tube to detect presence of CO_2 during exhalation.

The CO_2 detection device contains a chemical that changes color in the presence of carbon dioxide. After intubation, the detection device is attached to the endotracheal tube and the color on the device is observed. If the color turns from purple to yellow, it is an indication of successful endotracheal intubation since expired air from the lungs contains approximately 5% carbon dioxide.

If the patient is not breathing spontaneously, bilateral breath sounds should be checked by manual ventilation with a resuscitation bag. The placement of the stethoscope diaphragm should be along the midaxillary line. When the ET tube is

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TABLE 6-6 Common Problems during Intubation

Problem (Potential Cause)	Solution
Difficult to put blade or tube in mouth (Improper head position)	Use "sniffing" position by (1) tilting head back slightly and (2) moving chin anteriorly.
Trauma to teeth and soft tissues (Improper use of handle and blade)	Open mouth wider.
Unable to see epiglottis, larynx, or vocal cords (Blade is inside esophagus)	Do not pivot on teeth to lift blade and tongue.
Unable to advance ET tube when straight blade is used (ET tube is blocked by the light bulb on the right side of straight blade)	Withdraw *curved* blade until it reaches the vallecula (between base of tongue and epiglottis). Withdraw *straight* blade until it reaches the epiglottis.
Esophageal intubation, vomiting, and aspiration (Inserting the ET tube into *any* "opening" hoping it is the tracheal opening)	Rotate blade slightly counterclockwise (top of handle to left) to move light bulb out of the way.
Arrhythmias (Hypoxia caused by prolonged intubation attempt)	Find vocal cords and insert the ET tube through the cords under direct vision. Stop intubation. Ventilate and oxygenate.

properly placed (about 1.5 in. above the **carina**) the chest should expand and the abdomen should not have a gurgling sound during manual ventilation. Breath sounds heard on one side of the chest may suggest main-stem intubation. In the absence of obvious lung pathology (e.g., atelectasis), borderline main-stem intubation produces uneven bilateral breath sounds.

Finally, the depth of the ET tube should be checked with a chest radiograph. (Note: The chest radiograph is not done to confirm placement of the ET tube in the trachea.) For adult patients, the depth of intubation may be adjusted according to the chest radiograph. The tip of the ET tube should be about 1.5 in. above the carina if the patient's head is in the neutral position. Flexion of the head and neck can cause a 2 cm downward movement of the ET tube. Extension of the head and neck can move the tube upward by 2 cm (Godoy et al., 2012).

carina: The point at the lower end of the trachea separating openings of the main-stem bronchi.

✓ Do not check breath sounds at anterior chest locations close to the trachea since airflow in the *esophagus* can give false "breath sounds" in neonates and thin adults.

✓ For adult patients, the tip of an ET tube should be about 1.5 in. above the carina.

✓ In the absence of obvious lung pathology, uneven bilateral breath sounds may suggest main-stem intubation.

Signs of Esophageal Intubation

Placing an ET tube into the esophagus is a grave error. Hypoventilation, tissue and cerebral hypoxia are certain and immediate following esophageal intubation of an apneic patient. Furthermore, manual ventilation via an ET tube that has been placed in the esophagus may lead to aspiration of stomach contents and make subsequent intubations extremely difficult. Signs of esophageal intubation include

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oxygen desaturation (↓SpO_2), deteriorating vital signs, cyanosis, stomach distention, and aspiration.

✓ Esophageal intubation can be avoided by inserting the ET tube through the vocal cords under *direct vision*.

In almost all instances, esophageal intubation can be avoided by confirming that the ET tube passes through the vocal cords under *direct vision*. If the vocal cords are invisible or cannot be positively identified, the ET tube must not be inserted. Another experienced physician or practitioner should attempt to reintubate. Valuable time must not be wasted when a difficult intubation is encountered.

Esophageal detection device. The esophageal detection device (EDD) is a simple tool (e.g., esophageal syringe or bulb) to detect esophageal intubation in an emergency setting (Kasper & Deem, 1998). This device (e.g., bulb) provides a negative-pressure test using a compressible and self-inflating bulb with two openings. The upper end contains a one-way valve to allow air to escape from the bulb. The lower end has an adaptor that connects to the endotracheal tube. After intubation, the bulb is attached to the endotracheal tube and then compressed. With tracheal placement, the bulb draws air from the trachea and should reinflate to its original shape within 10 sec. With esophageal placement, the bulb receives little or no air from the constricted esophagus and it remains deflated (Wilkins et al., 2003). A false negative may occur if the tube is in the esophagus and the stomach is full of air. The EDD test results must match the patient's clinical signs following an intubation attempt.

RAPID SEQUENCE INTUBATION

rapid sequence intubation (RSI): Intubation with an endotracheal tube under controlled settings.

Rapid sequence intubation (RSI) describes an urgent need to gain control of a patient's airway. It has been done safely and successfully in both adult and pediatric patients (Davis et al., 2002; Dufour et al., 1995; Sagarin et al., 2002). RSI is done using an endotracheal tube under controlled settings to optimize the intubation conditions, to protect the airway against aspiration, and to facilitate ventilation and oxygenation.

Indications and Contraindications

Indications for rapid sequence intubation include airway obstruction, severe brain injury, severe hypoxemia, abnormal respiratory frequency, and hemodynamic instability (Table 6-7).

RSI should not be performed if a patient is able to sustain adequate ventilation and oxygenation while breathing spontaneously. It should not be attempted if the provider is unfamiliar with the proper procedure, intubation supplies, and drugs used in RSI. Following unsuccessful RSI attempts, surgical approach should be considered in special circumstances such as airway trauma or difficult anatomy.

Practice Guidelines

The procedure for RSI varies among different protocols. However, each protocol typically consists of the following elements: preparation, pre-RSI medications,

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TABLE 6-7 Indications for Rapid Sequence Intubation

Indication	Notes
Airway obstruction	Inability to maintain patent airway with other devices (e.g., oropharyngeal airway, laryngeal mask airway)
Severe brain injury	Glasgow coma scale of 8 or less
Severe hypoxemia	PaO_2/F_IO_2 (P/F) ratio < 250 mm Hg
Abnormal respiratory frequency	Spontaneous frequency < 10/ min or $>$ 30/min
Hemodynamic instability	Deteriorating hemodynamic values (e.g., vital signs, CVP, PAP, PCWP)

Modified from http://www.traumaburn.com. Retrieved April 21, 2004.

cricoid pressure, intubation, and post-RSI stabilization (Figure 6-13). (Bergen et al., 1997; Robinson et al., 2001; Sokolove et al., 2000; Smith et al., 2000).

In preparing for RSI, the following equipment and supplies should be readily available: cardiac monitor, intravenous access for pre-RSI medications, pulse oximeter, oxygen, drugs for advanced cardiovascular life support (ACLS), and cricothyrotomy tray for unsuccessful RSI attempts.

Sedation and muscle paralysis facilitate RSI. Different drugs are available for RSI, and they should be chosen based on the patient's condition, indications, and contraindications. Common pre-RSI medications include etomidate (Amidate) for sedation and induction (Guldner et al., 2003; Smith et al., 2000) and succinylcholine (Stewart, 2003; Walker, 1993) as a paralytic agent. For adult patients, 20 mg or 0.3 mg/kg of etomidate may be given intravenously over 30 to 60 sec. Succinylcholine may not be necessary if etomidate alone provides adequate sedation and results in successful intubation. If succinylcholine is needed, 100 mg or 1 to 1.5 mg/kg should be adequate. Since the onset of etomidate and succinylcholine is about 60 sec, intubation should be ready to proceed prior to administration of pre-RSI medications. Cricoid pressure using the Sellick's maneuver (Figure 6-14) may be done to close off the esophagus and to minimize aspiration. However, extreme care must be exercised because excessive cricoid pressure may also close off the airway itself (Walters, 2011).

✓ Pre-RSI medications should be chosen based on the patient's condition, indications, and contraindications.

✓ Common pre-RSI medications for adults include 20 mg of etomidate (Amidate) for sedation and 100 mg of succinylcholine.

✓ Cricoid pressure is applied to close off the esophagus and to minimize aspiration.

After sedation and relaxation of respiratory muscles are achieved, oral intubation is done using traditional method as described earlier in this chapter. If post-RSI paralysis and sedation are desired, vecuronium bromide (Norcuron, a steroidal-based nondepolarizing neuromuscular blocking agent), diazepam (Valium, an antianxiety agent), and fentanyl (Sublimaze, a synthetic opiate analgesic) may be used. The suggested adult IV dosages for vecuronium, diazepam, and fentanyl are 0.1 mg/kg, 5 to 10 mg, and 200 μg, respectively.

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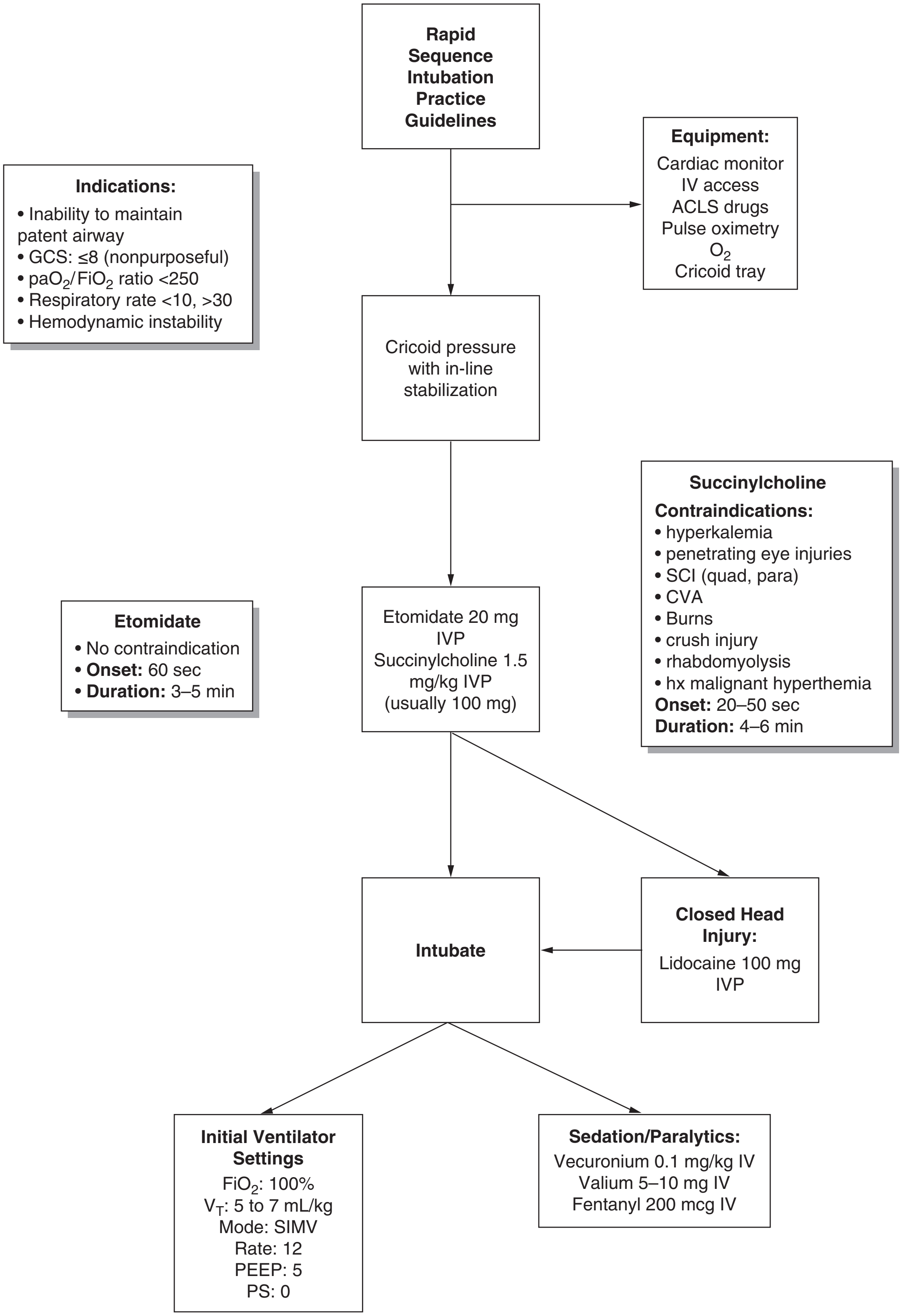
Rapid Sequence Intubation Practice Guidelines
Equipment:
Cardiac monitor
IV access
ACLS drugs
Pulse oximetry
O_2
Cricoid tray
Indications:
• Inability to maintain patent airway
• GCS: ≤8 (nonpurposeful)
• paO_2/FiO_2 ratio <250
• Respiratory rate <10, >30
• Hemodynamic instability
Cricoid pressure with in-line stabilization
Succinylcholine
Contraindications:
• hyperkalemia
• penetrating eye injuries
• SCI (quad, para)
• CVA
• Burns
• crush injury
• rhabdomyolysis
• hx malignant hyperthemia
Onset: 20–50 sec
Duration: 4–6 min
Etomidate
• No contraindication
• Onset: 60 sec
• Duration: 3–5 min
Etomidate 20 mg IVP
Succinylcholine 1.5 mg/kg IVP (usually 100 mg)
Intubate
Closed Head Injury:
Lidocaine 100 mg IVP
Initial Ventilator Settings
FiO_2: 100%
V_T: 5 to 7 mL/kg
Mode: SIMV
Rate: 12
PEEP: 5
PS: 0
Sedation/Paralytics:
Vecuronium 0.1 mg/kg IV
Valium 5–10 mg IV
Fentanyl 200 mcg IV

FIGURE 6-13 Rapid sequence intubation practice guideline.

Vanderbilt University Medical Center, Division of Trauma and Burns. Used with permission.

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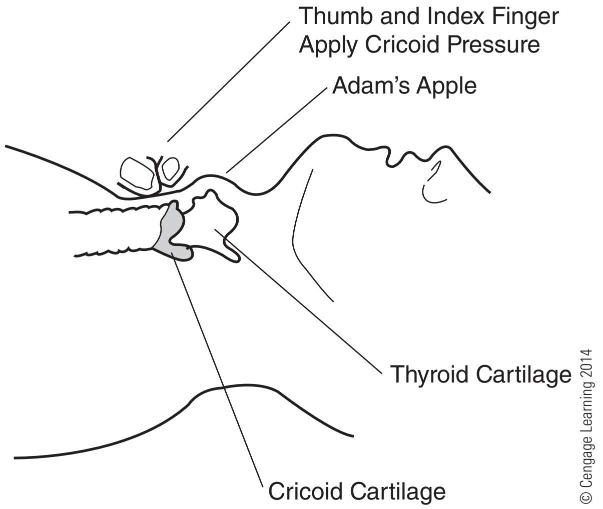

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FIGURE 6-14 Sellick's (cricoid pressure) maneuver.

MANAGEMENT OF ENDOTRACHEAL AND TRACHEOSTOMY TUBES

Once the patient is successfully intubated with an artificial airway, the airway must be managed properly to prevent complications. Failure to secure the airway may lead to inadvertent or self-extubation. Excessive cuff pressures may lead to tracheal mucosal tissue injuries. Finally, failure to humidify the secretions makes its removal from the artificial airway difficult and this condition may lead to pneumonia and atelectasis.

Securing Endotracheal and Tracheostomy Tubes

The ET tube can be secured by using adhesive tape or a commercially made harness. As shown in Figure 6-15A (oral intubation) and 6-15B (nasal intubation) they are used around the base of the head or neck for maximal security. Caution must be exercised as this technique may cause facial swelling and injuries to the lips when applied around the neck too tightly.

Since moisture often gathers between the tape and skin, tapes that can withstand moisture are more desirable. Zinc oxide base tape (by Hy-Tape International, New York) is one that sticks well to the skin when exposed to moisture.

Tracheostomy tubes are secured by tying a string to the two openings on the collar. The string goes around the neck for good fit and security.

Cuff Pressure

The estimated capillary perfusion pressure in the trachea is in the range of 25 mm Hg to 35 mm Hg. Lateral wall cuff pressure higher than this range can cause mucosal ischemia and tracheal wall tissue necrosis (Seegobin et al., 1984).

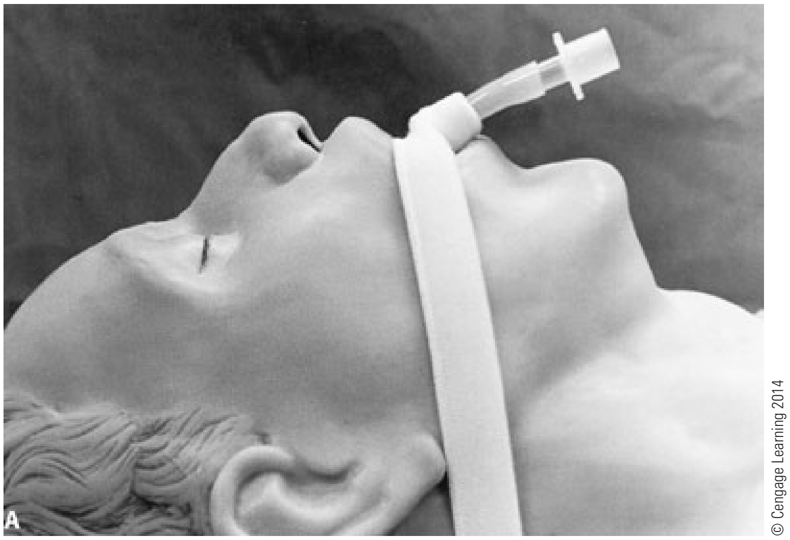

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FIGURE 6-15A Use of a commercially made harness to secure an oral endotracheal tube.

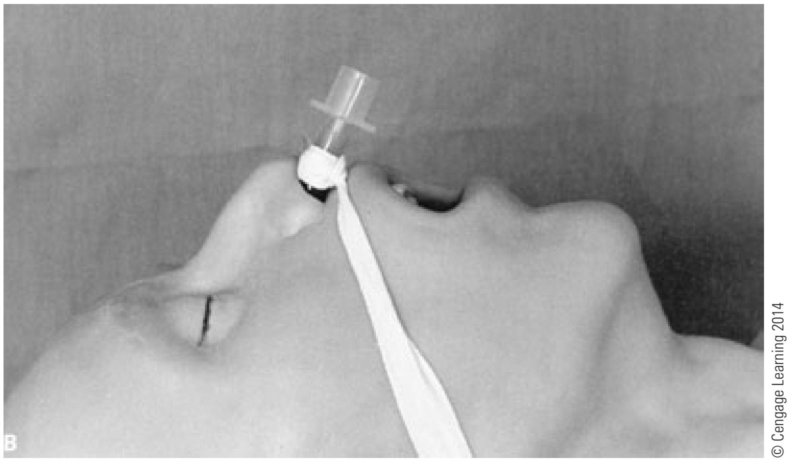

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FIGURE 6-15B One method of securing the tube of a nasally intubated patient.

✓ The ET tube cuff pressure should be 25 mm Hg or less to minimize pressure-induced injuries to the trachea.

The ET tube cuff pressure should be 25 mm Hg or less to allow adequate capillary perfusion in the trachea. For patients with hypotension, the cuff pressure should be kept even lower to compensate for the reduced capillary flow due to hypotension. Figure 6-16 shows the use of a pressure manometer and syringe to adjust the ET tube cuff pressure. Figure 6-17 shows a photograph of the Posey Cufflator with a built-in manometer. Air may be added to the ET tube cuff by pumping the bulb on the cufflator. Air may be removed from the ET tube cuff by opening the release valve on the cufflator.

Minimal Occlusion Volume and Minimal Leak Technique

If a cuff pressure manometer is not readily available, the minimal occlusion volume or minimal leak technique may be used to reduce the likelihood of pressure-induced injuries to the trachea caused by excessive cuff pressure.

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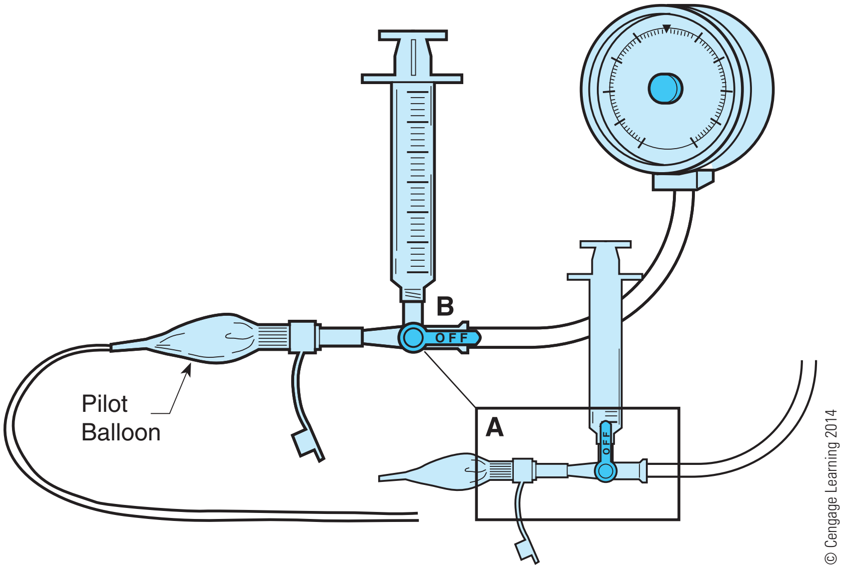

FIGURE 6-16 (A) When the stop cock points toward the syringe, the manometer measures the cuff pressure. (B) When the stop cock points toward the manometer, the syringe may be used to fill or withdraw air from the cuff.

✔ The minimal occlusion volume is obtained by inflating the cuff to a point at which no air leak is heard at end-inspiration.

✔ The minimal leak technique is done by inflating the cuff until the leak stops and then removing a small amount of air slowly until a *slight* audible leak can be heard at end-inspiration.

The minimal occlusion volume is obtained by inflating the cuff slowly until reaching a point at which no air leak is heard at end-inspiration. The air leak around the cuff can be checked by placing the stethoscope diaphragm on the trachea, as close to the location of the cuff as possible. The end-inspiration point is used because the trachea reaches its maximal diameter at end-inspiration.

The minimal leak technique is done by inflating the cuff until the leak stops and then removing a small amount of air slowly until a *slight* audible leak can be heard at end-inspiration (Chang, 1995).

Endotracheal Suctioning

Intubated patients are at risk for secretion retention because the ET tube and ventilator attachments form a closed system and do not allow removal of secretions.

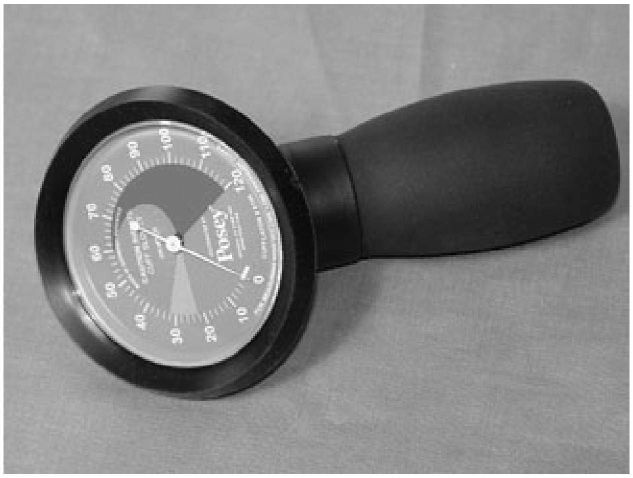

FIGURE 6-17 The Posey Cufflator.

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✓ The appropriate suction catheter size in French (Fr) may be estimated by multiplying the ET tube size by 3, and then dividing by 2. The equation is Fr = (mm × 3) / 2.

✓ To avoid suction-induced hypoxemia, preoxygenate the patient and keep the duration of suction to less than 15 sec.

Secretions must be removed by way of ET suctioning. However, frequent and inappropriate ET suctioning can cause mucosal damage and increase the incidence of suction-induced hypoxemia and arrhythmias. Therefore, ET suctioning should not be done on a preset sechedule; it should be done only when indicated.

The size of a suction catheter should be large enough for removal of secretions. However, an oversized suction catheter may impede airflow through the ET tube. As a rule of thumb, the outside diameter of the suction catheter should not exceed 50% of the inside diameter of the ET tube (Wilkins et al., 2003). For example, a size 12 Fr suction catheter should be used to suction a size 8 ET tube. The equation is (8 mm × 3)/2. The 3 in the equation is the factor to convert mm to Fr. The 2 in the equation is to obtain 50% of the ET tube diameter. If the calculate size is not available, the next *larger* suction catheter may be used.

The level of vacuum pressure should be kept between 70 and 150 mm Hg for adults and lower for infants and children (Figure 6-18). The effectiveness of suction (based on the amount of secretion removed) is optimal at a vacuum pressure of 100 mm Hg. Pressures higher than 100 mm Hg are not more effective but more likely to cause damage to the tracheal wall (Hahn, 2010).

Suction-induced hypoxemia may be avoided by preoxygenating the patient with 100% oxygen and limiting the duration of suctioning from 10 to 15 sec for adults and up to 5 sec for pediatric patients (Hahn, 2010). A closed in-line suction system (e.g., Ballard) may be useful to suction patients who have copious amounts of secretions. This system allows suctioning without the need to disconnect the ventilator circuit. Since it is a closed system, the PEEP level as well as other ventilation and oxygenation settings on the ventilator may be maintained throughout the suctioning period (Rodenhizer, 2004). As an added financial benefit over the traditional disposable suction catheters, the in-line suction system does not require frequent changes (Kollef et al., 1997).

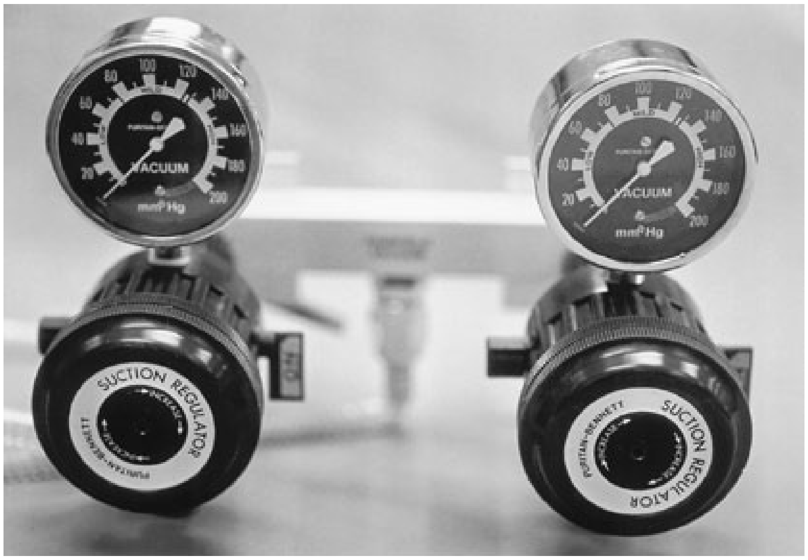

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FIGURE 6-18 A Puritan-Bennett suction regulator with vacuum pressures ranging from 0 to 200 mm Hg.

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Adult endotracheal tubes with a small dorsal lumen above the cuff are available to allow removal of subglottic secretions with a vacuum pressure of 20 mm Hg or less. Since the vacuum pressure is relatively low, obstruction or blockage of the dorsal lumen may occur. Patency of this lumen may be restored by injecting a small amount of air through the lumen (Tyco Healthcare, 2004). In one study, intermittent drainage of subglottic secretion has been shown to reduce the incidence of ventilator-associated pneumonia in patients receiving mechanical ventilation (Smulders et al., 2002).

The sequence outlined in Table 6-8 provides a general procedure for ET suctioning. It should be modified to suit individual situations and to comply with existing protocols. For example, routine irrigation of the ET tube saline solution before suctioning is not recommended (Demaray, 2002). Furthermore, use of saline during endotracheal suctioning procedure may cause dislodging of bacteria into the lower airway (Hagler et al., 1994).

Endotracheal Tube Changer

Occasionally, an ET tube may need to be changed (e.g., persistent cuff leak, tube too small). The flexible fiberoptic bronchoscope has been used successfully to change an ET tube without reintubation (Rosenbaum et al., 1981). Alternatively, an ET tube changer can be used to replace an existing endotracheal tube without the need to perform traditional intubation. A tube changer is a flexible guide that is ET tube size-specific and it should be sterilized before use. Some tube changers allow ventilation and oxygenation and they are ideal for extubation trial.

The main steps of using the endotracheal tube changer involve inserting the tube changer into the existing ET tube, deflating the cuff, stabilizing the changer and ET tube while removing the ET tube, replacing it with another ET tube, and inflating the cuff.

The distal end of the tube changer placed in the existing ET tube should be near the distal end of the ET tube. It should not protrude beyond the distal end of the ET tube. The proper placement of the tube changer can be determined by using the guide marks on the tube changer. Another approach is to insert the tube changer into a new identical ET tube until the distal end of the tube changer rests at the distal end of the new ET tube. The marker on the tube changer at the proximal end of the ET tube can then be noted and used as a visual guide.

✓ Use of tube changers can lead to complications that include laceration of lateral wall, bronchial perforation with pneumothorax, loss of airway, hypoxemia, and potential need of a surgical airway.

✓ For safety reasons, the person using the ET tube changer must be proficient in intubation and experienced in airway management.

Stabilization of the tube changer during the entire procedure is probably the most crucial step. An unstable tube changer during the procedure may lead to main-stem intubation, esophageal intubation, or lung laceration (DeLima et al., 1991). Use of tube changers can lead to complications that include laceration of lateral wall, bronchial perforation with pneumothorax, loss of airway, hypoxemia, and potential need of a surgical airway (Nates et al., 2001). For safety reasons, the person using the ET tube changer must be proficient in intubation and experienced in airway management.

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TABLE 6-8 Endotracheal Suctioning

Procedure	Rationale
Wash hands and gather all suction supplies (catheter, sterile gloves, water, water container, and saline solution).	Avoid need to obtain other supplies once sterile gloves have been put on hands.
Explain procedure to patient.	Assure patient understanding and cooperation.
Adjust vacuum to 100 mm Hg.	Prevent excessive vacuum and mucosal damage.
Put sterile water in container.	For testing suction device and flushing secretions inside catheter.
Put on sterile gloves using aseptic technique.	Minimize nosocomial infection.
Designate "sterile" and "contaminated" hands.	Use sterile hand to handle all supplies requiring aseptic technique (i.e., suction catheter). Use contaminated hand to handle all other supplies (e.g., ET tube adaptor, suction tubing).
Attach suction catheter (sterile hand) to suction tubing (contaminated hand).	Ensure sterile and aseptic techniques.
Test vacuum and suction with sterile water.	Ensure proper function of suction setup.
Remove ET tube adaptor and irrigate with 5 mL of sterile saline or mucolytic agent only if indicated (contaminated hand).	Loosen secretions.
Manually hyperinflate the patient's lungs with resuscitation bag.	Ensure adequate ventilation and oxygenation. Seek help if necessary.
Insert catheter into ET tube (sterile hand) and advance until resistance is met. Withdraw catheter slightly.	Avoid suctioning the tracheal wall and minimize mucosal damage.
Activate suction (contaminated hand) and withdraw catheter (sterile hand) by rotating the catheter.	Increase removal of secretions.
Limit the duration of suction from 10 to 15 sec for adults (5 sec for children)	Prevent suction-induced hypoxia and arrhythmias.
Auscultate chest and repeat suction if necessary.	Avoid unnecessary suctioning.

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SPEAKING VALVES

Fenestrated tracheostomy tubes have been in use for many years to facilitate weaning of tracheostomy tube and to allow phonation (Mallinckrodt, 2011). With the airway opening of a fenestrated tracheostomy tube blocked or plugged, the small fenestrated openings along the body of the tube allow the patient to move air through the vocal cords making phonation possible (Figure 6-19).

speaking valve: A one-way valve attachment to the tracheostomy tube that allows the user to talk.

✓ Speaking valves block the exhaled air through the tracheostomy tube and direct it through the vocal cords making phonation possible.

A **speaking valve** is a device which consists of a one-way valve that fits on a traditional or fenestrated tracheostomy tube over a 15-mm connection. The one-way valve allows the patient to breathe in, but exhaled air is blocked and diverted around the deflated cuff or fenestrations. The air moves through the vocal cords making phonation possible (Figure 6-20). Some common speaking valves are the Passy-Muir (Passy-Muir Inc., Irvine, CA), the Phonate (Mallinckrodt, 2011), and the Montgomery and Ventrach speaking valves (Boston Medical Products, 2012).

Contraindications

✓ Contraindications for using speaking valve include laryngeal stenosis, vocal cord paralysis, and severe tracheal stenosis.

Contraindications for using a speaking valve include laryngeal stenosis, vocal cord paralysis, and severe tracheal stenosis. These conditions impede airflow making ventilation and phonation difficult (Mallinckrodt, 2011).

Safety Requirements

✓ The design of the speaking valve allows only inhalation through the tracheostomy tube, but not exhalation.

Before the speaking valve is placed on a cuffed tracheostomy tube, the cuff must first be deflated. An inflated cuff would make exhalation through the tracheostomy tube impossible because the design of the speaking valve allows only inhalation, but not exhalation. A deflated cuff would allow the patient to exhale around the tracheostomy tube, through the vocal cords and the mouth or nose.

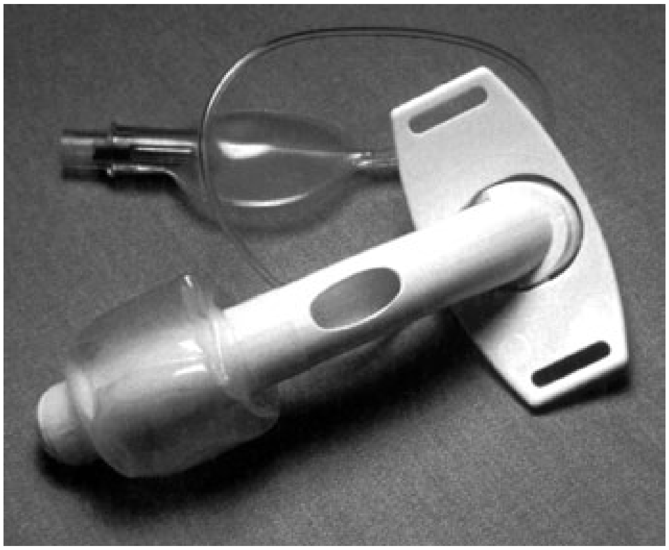

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FIGURE 6-19 A fenestrated tracheostomy tube.

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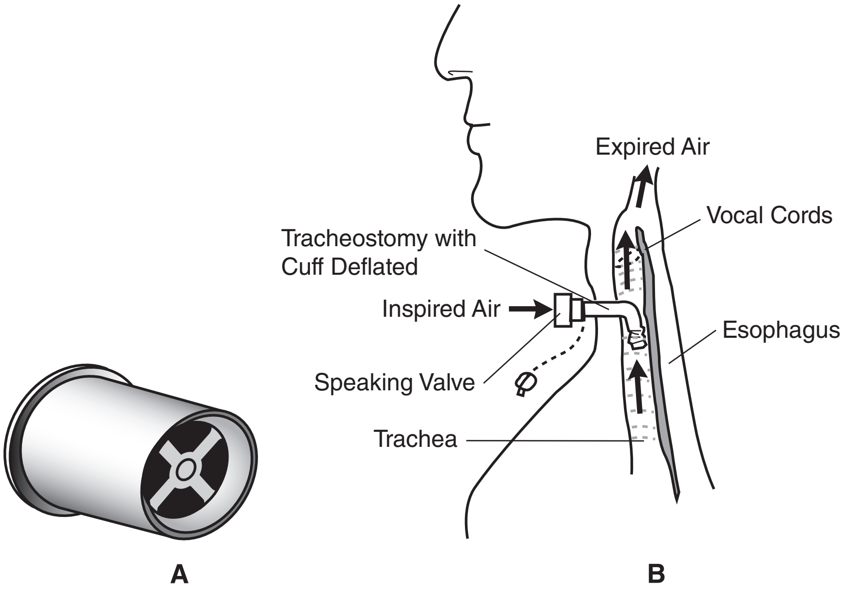

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FIGURE 6-20 (A) Speaking valve. (B) The one-way valve dose not allow exhalation through the tracheostomy tube. The exhaled air is directed toward the vocal cords making phonation possible.

✔ Before the speaking valve is placed on a fenestrated tracheostomy tube, the fenestrations must be opened by removing the nonfenestrated inner cannula or using a fenestrated inner cannula.

Before the speaking valve is placed on a fenestrated tracheostomy tube, the fenestrations must be opened by removing the nonfenestrated inner cannula or using a fenestrated inner cannula. The cuff of the fenestrated tracheostomy tube may be inflated or deflated. This technique allows the exhaled air to move toward the vocal cords and the mouth or nose (Pruit, 2003). Table 6-9 summarizes the safety requirements in using a speaking valve on a tracheostomy tube.

Positive Pressure Ventilation

Air leak is common when using a speaking valve. For patients who are breathing spontaneously, the amount of air leak is of no concern. In mechanically ventilated patients, the air leak may require careful and periodical adjustments of the tidal volume, PEEP, and alarm settings (Pruitt, 2003). To ensure adequate ventilation and oxygenation, the vital signs and pulse oximetry of the patient must be closely

TABLE 6-9 Safety Requirements in Using a Speaking Valve

Type of Tracheostomy Tube	Requirement
Traditional	Cuff must be deflated.
Fenestrated	Fenestrations must be opened by: removing the nonfenestrated inner cannula, or using a fenestrated inner cannula. Cuff may be inflated only when fenestrations are open.

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monitored. Deteriorating vital signs and oxygen desaturation are signs of speaking valve malfunction, severe airflow obstruction, or air leak.

EXTUBATION

Extubation should be done as soon as feasible. Early extubation not only provides immediate relief to the patient, but also shortens the duration of a hospital stay, reduces health care costs, and conserves resources (Cheng, 1995; Lichtenthal et al., 1995; Velasco, 1995). In one study of patients undergoing coronary artery bypass grafting, the average saving per patient was $6,000 in the early extubation group (Arom, 1995).

Predictors of Successful Extubation

A patient is ready for extubation after regaining airway reflexes and showing no signs of cardiopulmonary distress. Strong productive coughs, small amount of secretions, and hemoglobin level >10g/dL are good predictors of successful extubation (Khamiees, 2001). Other objective criteria for assessing a patient's readiness for extubation include the rapid shallow breathing index, blood gases, muscle strength, and general cardiopulmonary signs.

✓ The patient should be allowed to breathe spontaneously for at least three minutes before taking measurements. Otherwise, the f/V_T index may not reflect the patient's actual condition.

Rapid shallow breathing index. The rapid shallow breathing (f/V_T) index can be obtained easily by measuring the breathing frequency and minute volume during 1 min of spontaneous breathing (Epstein, 1995; Yang et al., 1991). f/V_T is calculated by dividing the spontaneous breathing frequency per minute by the average tidal volume in liters. A value of less than 100/min/L is highly predictive of successful extubation outcome.

✓ Competent personnel and intubation supplies must be readily available during extubation.

Other common indicators. Acceptable blood gases, ventilatory reserve, and general cardiopulmonary signs, infrequent need for suctioning (>4 hours), being alert, and SpO_2 >95% are other useful indicators that may be used to guide the extubation decision (Pronovost et al., 2002). These criteria and the rapid shallow breathing index are very simple and easy to use. They are summarized in Table 6-10 along with their respective methods of assessment.

Procedure

✓ The person who is doing the extubation must be proficient in intubation.

There should be no disagreement that extubation is easier than intubation. Nevertheless, the person who is doing the extubation must be proficient in intubation as well. Since the criteria used for the extubation decision cannot predict a successful outcome every time, one must anticipate the need for reintubation on short notice. Intubation supplies must also be readily available during extubation.

Before extubation, the procedure is explained to the patient and the patient is positioned in a Fowler's (semi-sitting) position. Hyperinflation and oxygenation are provided to the patient with a manual resuscitator via the ET tube. The ET tube is then suctioned.

TABLE 6-10 General Criteria for Extubation

Criteria	Methods of Assessment
Rapid shallow breathing index	f/V_T less than 100/min/L
Blood gases	Acceptable blood gases on F_IO_2 less than 40% and spontaneous minute ventilation less than 10 L/min PaO_2/F_IO_2 (P/F ratio) $>$ 250 mm Hg
Ventilatory reserve	Maximal inspiratory pressure > -20 cm H_2O Vital capacity $>$ 15 mL/kg
Cardiopulmonary assessment	Absence of cardiopulmonary problems (e.g., CHF, pulmonary edema, pneumonia, tachycardia, arrhythmia, chest retractions, distended stomach)

(Data from Epstein, 1995; Listello et al., 1994; Whelan et al., 1994; White, 2002; Whitten, 1997.)

If the patient has an adequate vital capacity and cough reflex, the cuff is deflated completely and the ET tube is removed. Encourage the patient to breathe deeply and cough, and use a Yankaur (rigid tonsil tip) suction device to remove excess secretions in the patient's oropharynx.

If the patient does not have an adequate vital capacity and cough reflex, the suction catheter may be left extending from the distal end of the ET tube and continuous suction is applied while the ET tube is being removed. An alternate method is to leave the suction catheter in the oropharynx with continuous suction while the ET tube is being removed. Either method may be used to remove secretions from the patient's airway or oropharynx (Figure 6-21).

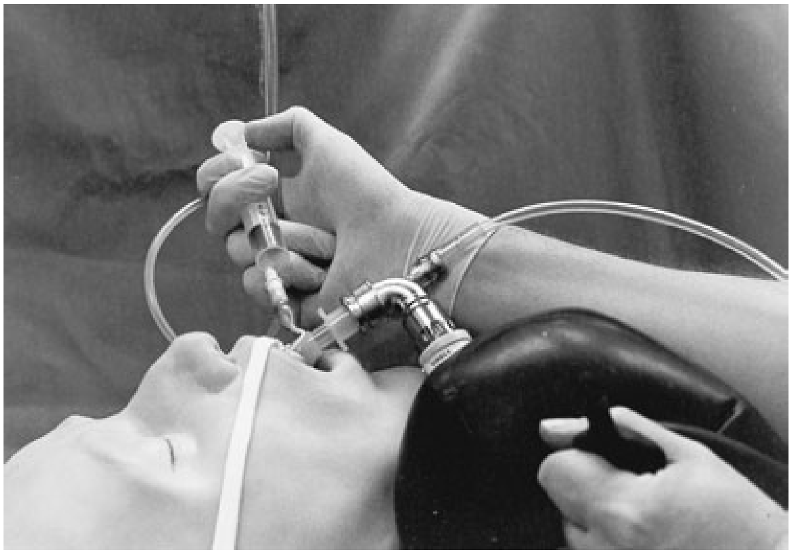

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FIGURE 6-21 Use of a flow-inflating manual resuscitator, suction catheter, and a syringe during extubation.

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✓ Aspiration, laryngospasm, hoarseness, and laryngeal and subglottic edema are some complications immediately after extubation.

Vital signs, blood gases, and signs of tissue damage should be assessed carefully after extubation. Some immediate postextubation complications include aspiration, laryngospasm, hoarseness, and laryngeal and subglottic edema. Other more severe complications that may not be immediately apparent are mucosal injuries, laryngeal stenosis, tracheal inflammation, dilation or stenosis, and vocal cord paralysis (Young et al., 1995).

Unplanned Extubation

unplanned extubation: Unexpected removal of an endotracheal or tracheostomy tube before the patient is ready for extubation.

Unplanned extubation or inadvertent extubation (self-inflicted or accidental) accounts for about 8% to 10% of all extubations in ICU patients (Listello et al., 1994). For patients who self-extubate the endotracheal tube, about 50% of them do not need to be reintubated (Betbese et al., 1998; Chevron et al., 1998). Whether or not to reintubate the patient can be a difficult decision. Delayed reintubation may lead to adverse outcomes such as hypoventilation, hypoxemia, and hypoxia. In general, the decision to reintubate may be based on clinical observations and the criteria for extubation (i.e., rapid shallow breathing index, blood gases, ventilatory reserve, and general cardiopulmonary signs). However, these measurements may not have been done immediately before extubation since the extubation is not planned.

To avoid this problem, other criteria based on routinely available patient information have been identified and used for the reintubation decision. They are summarized in Table 6-11. In the model set, the presence of four or more factors indicates the need for reintubation and the presence of three or fewer factors reflects a satisfactory patient outcome without reintubation (Listello et al., 1994).

TABLE 6-11 Clinical Predictors for Reintubation

Unfavorable Clinical Predictor*	Rationales
1. SIMV or AC frequency > 6/min	Patient is dependent on the ventilator.
2. Most recent pH ≥ 7.45	Oxyhemoglobin saturation curve shifts to left (↑O_2 affinity and ↓O_2 release to tissues).
3. Most recent $PaO_2/F_IO_2 < 250$ mm Hg	Poor oxygenation status.
4. Highest heart rate in the past 24° > 120/min	Cardiac compensation for poor perfusion or oxygenation.
5. Presence of ≥ 3 medical disorders	Potential of medical complications.
6. Not alert	Poor mental status; blunted drive for breathing.
7. Reason for intubation other than preoperative	Presence of medical problems and potential complications.

Presence of four or more predictors favors reintubation. Presence of three or less predictors indicates no need for reintubation.

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COMPLICATIONS OF ENDOTRACHEAL AIRWAY

Endotracheal intubation is an extremely useful procedure in the establishment of an artificial airway but it also carries many potential complications. As shown in Table 6-12, complications may develop at different stages of intubation and extubation. Some conditions are life-threatening (e.g., esophageal intubation, bradycardia) while others are minor and often reversible (e.g., pressure sores, hoarseness). It is essential to understand and recognize the potential complications so that appropriate steps may be taken to avert harmful outcomes.

TABLE 6-12 Complications Related to Use of Endotracheal Tube

Sequence of Events	Complications
During intubation	Trauma to teeth and soft tissues Esophageal intubation Vomiting and aspiration Hypoxia due to prolonged intubation attempt Arrhythmias Bradycardia due to vagal stimulation
While intubated	Obstruction by secretions Pneumonia and atelectasis Kinking of ET tube Aspiration (from feeding and ineffective cuff) Mucosal injuries Laryngeal damage Improper tube position (too high, too low) Pressure sores around ET tube Inadvertent extubation Sinusitis (nasal intubation)
Immediately after extubation	Aspiration Laryngospasm Hoarseness Laryngeal and subglottic edema
Following extubation	Mucosal injuries Laryngeal stenosis Tracheal inflammation, dilation, stenosis Vocal cord paralysis

(Data from Chang, 1995; Dillier et al., 2004; White, 2002; Whitten, 1997; Young et al., 1995.)

During Intubation

Trauma to the teeth and soft tissues can occur during intubation.

Esophageal intubation is commonly done by inexperienced practitioners.

The ASA Task Force on the Management of the Difficult Airway recommends a limit of three intubation attempts to minimize patient injury.

vagus nerve: The pneumogastric or tenth cranial nerve. Its superior and recurrent laryngeal nerves and their branches adjoin the upper end of trachea and are sensitive to stimulation by the endotracheal tube or suction catheter.

Trauma to the teeth and soft tissues can occur during intubation since ET intubation is often done in emergency situations. Difficult intubations compound the problem and lead to injuries when the patient has one or more of these conditions: obesity, receding chin, overbite, rigid or short neck, or blood or vomitus in oropharynx.

Esophageal intubation is the most dangerous complication. It may occur when it is performed by an inexperienced practitioner or under awkward patient positions (e.g., patient lies on the floor). Esophageal intubation frequently leads to vomiting and aspiration, thus making subsequent intubation attempts more difficult or nearly impossible.

Prolonged or repeated intubation attempts lead to hypoxia and, if uncorrected, dangerous arrhythmias may occur. Excessive stimulation of the **vagus nerve** can cause bradycardia. Arrhythmias induced by hypoxia and bradycardia caused by vagal stimulation may be reversed by removing the ET tube and oxygenating the patient until a normal sinus rhythm returns. The ASA Task Force on the Management of the Difficult Airway recommends a limit of three intubation attempts to minimize patient injury (Mort, 2004).

While Intubated

Failure to remove retained secretions could lead to pneumonia and atelectasis.

For an orally intubated patient, the distance marking on the ET tube (e.g., 22 cm) is the distance from the distal end of the ET tube to the patient's lips or incisors.

Complications that occur while the patient is intubated vary greatly according to the duration of ET tube placement and the airway management techniques. As a general rule, the longer an ET tube is in place, the more likely that complications will occur while the patient is intubated.

Since normal mucocillary functions of the mucosal membrane and the cough reflex are lost with an ET tube in place, retention of secretions must be removed promptly. If secretions are thick, irrigation with saline solution or acetylcysteine (Mucomyst®) should be done before suctioning. Failure to remove retained secretions may lead to pneumonia and atelectasis (Chang, 1995).

Kinking of an ET tube may be corrected by repositioning the connection between the ET tube and the circuit. Inadvertent extubation may be prevented by properly sedating the patient or by using temporary restraints on the forearms.

The position of the ET tube should be checked frequently by chest auscultation and concurrently when a routine chest radiograph is done. Once the ET tube is properly placed, the distance marking on the ET tube (centimeter mark) should be noted on the ventilator flow sheet. This reference number provides a quick reference point but should not be used as a substitute for routine assessment of the ET tube position.

Immediately after Extubation

Extubation of a semiconscious patient may stimulate the vocal cords and lead to reflex spasm.

Laryngospasm usually occurs as a result of extubation when the patient is semiconscious. Extubation during this excitement stage tends to stimulate the vocal cords and lead to reflex protective spasm. For this reason, extubation should be done when the patient is either deeply anesthesized or, preferably, fully awake (Whitten, 1997).

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✓ Stridor is the harsh or high-pitched sound heard during spontaneous respiration and it is initially treated with racemic epinephrine.

Stridor is heard when laryngospasm or laryngeal and subglottic edema occur. In minor cases of stridor, use of a cool aerosol and 0.25 to 0.5 mL of 2.25% racemic epinephrine in 5 mL of saline may be helpful. Dexamethasone at 0.15 mg/kg may help prevent worsening laryngeal and subglottic edema. In severe cases of laryngospasm, airway obstruction may have developed and reintubation is often required (Whitten, 1997).

Following Extubation

Mucosal injuries, laryngeal stenosis, tracheal damages (inflammation, dilation, and stenosis), and vocal cord paralysis are some long-term complications following extubation. The best way to avoid these complications is to practice proper airway care while the patient is intubated.

SUMMARY

The most important element of airway management is intubation. Esophageal intubation can be deadly and it must not be done under any circumstance (e.g., secretions in the airway, poor view, or awkward patient position). The golden rule for endotracheal intubation is "If you don't see it (the vocal cords or related structures), don't do it."

Use of artificial airways also requires proper and routine maintenance to prevent complications. Humidification, suctioning, cuff pressure, and unplanned extubation are some important aspects in airway management during mechanical ventilation. Since the artificial airway is inside the patient's mouth and trachea and is not readily visible, practitioners must have a keen sense of observation and timely clinical judgment to detect any complications or potential troubles.

Self-Assessment Questions

1. The decision to intubate a patient is based on all of the following indications with the *exception* of:

A. airway obstruction.
B. hypoxemia.
C. mechanical ventilation.
D. removal of secretions.

2–5. Match the indications for using an artificial airway with the respective examples.

Indication	**Example**
2. Relief of airway obstruction	A. Absence of coordinated swallow
3. Protection of the airway	B. Ventilatory failure
4. Facilitation of suctioning	C. Excessive secretions
5. Support of ventilation	D. Epiglottitis

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6. The therapist is getting ready to intubate a patient who has been classified as Class 3 using the Mallampati method. The therapist should _______ because the _______ are seen during assessment.

   A. provide conscious sedation, soft palate and base of uvula
   B. provide conscious sedation, fauces, uvula, and tonsillar pillars
   C. seek anesthesia consultation, soft palate, and base of uvula
   D. seek anesthesia consultation, fauces, uvula, and tonsillar pillars

7. A therapist is preparing to intubate a 49-year-old, 60-kg (132-lb) patient, and two endotracheal tubes, size 7 and size 8 are available. The therapist should use the _______ tube because it offers _______ than the other ET tube.

   A. size 8, lower airflow resistance
   B. size 8, higher airflow resistance
   C. size 7, lower airflow resistance
   D. size 7, higher airflow resistance

8. The therapist is called to intubate a patient in the dialysis unit. Since there are no crash cart and intubation equipment nearby, the therapist is asked to bring along the minimal supplies needed for intubation. In addition to the laryngoscope handle, blade, and endotracheal tube, which of the following supplies is the most essential item for intubation and initiation of ventilation?

   A. water-soluble lubricant
   B. tape
   C. stylet
   D. 10-mL syringe

9. A therapist is performing an elective intubation on a 32-year-old, 78-kg (172-lb), 5 ft 2 in. patient in the intensive care unit. Based on the patient's physical characteristics, the therapist should use a size _______, _______ laryngoscope blade.

   A. 2, Miller
   B. 3, Miller
   C. 4, Macintosh
   D. 5, Macintosh

10. When the therapist intubates the patient in the preceding question, the therapist should place the tip of the blade just under the _______ and use the laryngoscope handle to _______.

    A. tongue, pry the mouth open
    B. tongue, lift up anteriorly
    C. epiglottis, pry the mouth open
    D. epiglottis, lift up anteriorly

11. During intubation of an apneic patient, the patient develops arrhythmias. The therapist should remove the blade and endotracheal tube immediately and provide:

    A. cardiac defibrillation and ventilation.
    B. 100% oxygen via a nonrebreathing ventilation.
    C. 100% oxygen via manual resuscitation bag/mask system.
    D. chest compression and mask.

12. The best approach to avoid esophageal intubation is to ensure that the endotracheal tube goes through the _______ under direct vision.

    A. underside of tongue
    B. pharynx
    C. vocal cords
    D. epiglottis

13. Trauma done to the teeth of a patient undergoing an intubation procedure is most likely caused by using:

    A. a straight blade.
    B. a curved blade.
    C. an oversized endotracheal tube.
    D. pivoting action to lift blade and tongue.

14. After intubating a spontaneously breathing 40-kg (88-lb) patient, all of the following signs may be used to assess correct placement of the endotracheal tube *except*:

    A. presence of breath sounds at the sternum.
    B. loss of speech.
    C. air movement over the ET tube opening.
    D. formation of moisture inside the ET tube.

15. The onset time for etomidate and succinylcholine used in rapid sequence intubation is about _______ min and these drugs last about _______ min.

    A. 1, 1
    B. 1, 5
    C. 5, 10
    D. 5, 20

16. In order to avoid mucosal injuries of the trachea, the cuff of an endotracheal tube may be managed by any one of the following techniques *except*:

    A. minimal occlusion volume.
    B. periodic deflation for 20 minutes q 4°.
    C. cuff pressure less than 25 cm H_2O.
    D. minimal leak technique.

17. A speaking valve attached to a cuffed tracheostomy tube does not allow the patient to _______ through the valve. For this reason, the cuff must be _______.

    A. exhale, inflated
    B. inhale and exhale, inflated
    C. exhale, deflated
    D. inhale and exhale, deflated

18. A physician asks the therapist to evaluate Mr. King, a postabdominal surgical patient, for possible extubation. Based on the information below, the therapist recommends extubation because the patient meets all of the criteria for extubation except:

    A. rapid shallow breathing (f/V_T) index of 150/min/L.
    B. PaO_2/F_IO_2 of 280 mm Hg.
    C. maximal inspiratory pressure of –38 cm H_2O.
    D. F_IO_2 of 35%.

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19. A 25-year-old postappendectomy patient extubated herself and the therapist is asked to evaluate this patient for possible reintubation. Based on the following list of clinical predictors for reintubation, the therapist should recommend that the patient _______ since she has met _______ of the predictors.

Unfavorable Clinical Predictor
1. SIMV frequency = 8/min
2. Most recent pH = 7.42
3. Most recent PaO_2/FIO_2 = 210 mm Hg
4. Highest heart rate in the past 24° = 110/min
5. Patient diagnosis is appendicitis
6. Patient is alert
7. Patient is in postoperative recovery

A. be reintubated, 2
B. be reintubated, 4
C. not be reintubated, 2
D. not be reintubated, 4

20. A patient develops bradycardia during an intubation attempt. The therapist should stop intubation immediately and _______ the patient since this condition is likely caused by _______.

A. rest, vagal stimulation
B. ventilate, hypoventilation
C. oxygenate, hypoxia
D. defibrillate, impending cardiac arrest

21. Several hours after endotracheal intubation, a 35-year-old patient becomes restless in spite of using sedatives and analgesics. The physician is concerned that the ET tube may move down the main bronchus because of excessive head movement. The therapist should recommend _______ and the tip of the ET tube be positioned about _______ in. from the carina.

A. auscultation, 0.5
B. auscultation, 1.5
C. chest radiograph, 0.5
D. chest radiograph, 1.5

Answers to Self-Assessment Questions

1. B.
2. D.
3. A.
4. C.
5. B.
6. C.
7. A.
8. D.
9. B.
10. D.
11. C.
12. C.
13. D.
14. A.
15. B.
16. B.
17. C.
18. A.
19. C.
20. A.
21. D.

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Chapter 7

Noninvasive Positive Pressure Ventilation

David W. Chang

Outline

- Introduction
- Terminology
- Physiologic Effects of NPPV
- Use of Continuous Positive Airway Pressure (CPAP)
  - *Obstructive Sleep Apnea*
- Use of Bilevel Positive Airway Pressure (Bilevel PAP)
- Common Interfaces for CPAP and Bilevel PAP
  - *Nasal Mask*
  - *Oronasal Mask*
  - *Nasal Pillows*
  - *Full-Face Mask*
- Potential Problems with Interfaces
- Titration of Continuous Positive Airway Pressure
  - *Autotitration*
  - *Ramp*
  - *C-Flex™*
- Titration of Bilevel Positive Airway Pressures
  - *Bi-Flex™*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

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Key Terms

- apnea index
- apnea-hypopnea index
- bilevel positive airway pressure (bilevel PAP)
- continuous positive airway pressure (CPAP)
- desaturation index
- expiratory positive airway pressure (EPAP)
- full-face mask
- hypopnea
- inspiratory positive airway pressure (IPAP)
- nasal mask
- nasal pillows
- noninvasive positive pressure ventilation (NPPV)
- obstructive sleep apnea (OSA)
- oronasal mask
- positive end-expiratory pressure (PEEP)

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- List the physiologic effects of NPPV.
- Describe the clinical application of continuous positive airway pressure (CPAP).
- Outline the signs of obstructive sleep apnea and its management.
- Discuss the use of Bilevel Positive Airway Pressure (bilevel PAP).
- Describe the characteristics of the common interfaces for CPAP and bilevel PAP.
- List the clinical application and potential problems with nasal mask, oronasal mask, nasal pillows, and full-face mask.
- Describe the procedure to set up and implement CPAP and bilevel PAP.

INTRODUCTION

noninvasive positive pressure ventilation (NPPV): NPPV provides assisted ventilation without an artificial airway.

Noninvasive positive pressure ventilation (NPPV) is a technique of providing ventilation without the use of an artificial airway. It has been used successfully in the management of airflow obstruction in sleep apnea (Guilleminault, et al. 1998) and in the reduction of respiratory workload in gross obesity (Pankow et al., 1997). More recently NPPV has been used in the management of acute respiratory failure. In selected patients with acute ventilatory failure, NPPV is as effective as conventional mechanical ventilation in improving gas exchange (Abou-Shala & Meduri, 1996). Since an artificial airway is not required in the use of NPPV, fewer

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complications and a shorter stay in an acute care setting are two additional benefits of NPPV (Antonelli et al., 1998).

During the polio epidemics, negative pressure ventilators (i.e., iron lungs) were used to provide ventilation by generating a pressure gradient between the atmosphere and lungs. Air flows into the lungs and ventilation occurs when the pressure in the lungs becomes subatmospheric. Disadvantages of negative pressure ventilators include upper airway obstruction and lack of access for patient care. Modern ventilators generate the pressure gradient between the atmosphere and lungs by positive pressure. With positive pressure ventilation, the pressure in the airway opening is higher than that in the lungs. Some of the disadvantages of positive pressure ventilation are tracheal injury, infection, ventilator-associated pneumonia, barotrauma, and prolonged hospital stay (Antonelli et al., 1998; Diaz et al., 1997; Keenan et al., 1997; Kramer et al., 1995).

NPPV provides ventilation via the patient's nose, mouth, or face (nose and mouth) without an artificial airway. For this reason, many complications of negative and positive pressure ventilation are minimized or eliminated.

✓ With NPPV, many complications of negative and positive pressure ventilation are minimized or eliminated.

TERMINOLOGY

In noninvasive positive pressure ventilation, the meanings of some terms are slightly different from traditional usage. These terms, abbreviations, and a brief description for each term are outlined in Table 7-1.

PHYSIOLOGIC EFFECTS OF NPPV

As in traditional positive pressure ventilation, NPPV has two primary pressure settings. One pressure setting is used during the inspiratory phase and the other during the expiratory phase.

During the inspiratory phase, the **inspiratory positive airway pressure (IPAP)** works like any other positive pressure breathing (ventilation) device. The IPAP level is similar to the peak airway pressure in traditional mechanical ventilation. In general, the set value is directly related to the IPAP level. A higher IPAP level would result in a larger tidal volume and minute ventilation. The **expiratory positive airway pressure (EPAP)** is the same as PEEP during mechanical ventilation or CPAP during spontaneous breathing. In addition to its ability to improve oxygenation by increasing the functional residual capacity, EPAP also relieves upper airway obstruction with its splinting action.

inspiratory positive airway pressure (IPAP): An airway pressure that is above 0 cm H_2O during the inspiratory phase of a respiratory cycle.

expiratory positive airway pressure (EPAP): An airway pressure that is above 0 cm H_2O during the expiratory phase of a respiratory cycle.

The level of IPAP and EPAP can be titrated according to a patient's oxygenation and ventilation needs. Since two benefits of NPPV are improvement of PO_2 and PCO_2 (Brown et al., 1998; Nicholson et al., 1998), these two parameters can be used as titration endpoints. If arterial blood gas results are available, oxygenation (PaO_2) and ventilation ($PaCO_2$) endpoints can be easily assessed. Alternatively, pulse oximetry (SpO_2) and capnography ($P_{ET}CO_2$) may be used for the titration of appropriate IPAP and EPAP levels.

✓ PaO_2, $PaCO_2$, SpO_2, and $P_{ET}CO_2$ may be used for titration of IPAP and EPAP.

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TABLE 7-1 Terms Used in NPPV

Abbreviation	Term	Notes
NPPV	Noninvasive positive pressure ventilation	1. Ventilation without an artificial airway 2. May be used as CPAP or bilevel PAP
CPAP	Continuous positive airway pressure	1. Positive airway pressure during spontaneous breaths 2. No mechanical breaths 3. CPAP is active when IPAP = EPAP
Bilevel PAP	Bilevel positive airway pressure	1. Provides IPAP and EPAP 2. CPAP is active when IPAP = EPAP 3. Also known as BiPAP™
IPAP	Inspiratory positive airway pressure	1. Controls peak inspiratory pressure during inspiration
EPAP	Expiratory positive airway pressure	1. Controls end-expiratory pressure 2. Used as CPAP when IPAP = EPAP 3. Used as PEEP when IPAP > EPAP
PEEP	Positive end-expiratory pressure	1. Positive airway pressure at end expiratory phase 2. With mechanical breaths
PS	Pressure Support	1. Positive pressure plateau to augment spontaneous V_T
PCV	Pressure-controlled ventilation	1. Ventilation provided by a preset peak inspiratory pressure
PPV	Positive pressure ventilation	1. Ventilation provided by a positive airway pressure

USE OF CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

continuous positive airway pressure (CPAP): CPAP does not include any mechanical breaths.

Continuous positive airway pressure (CPAP) provides positive airway pressure during spontaneous breaths and it does not include any mechanical breaths. For this reason, the work of breathing is entirely assumed by the patient. CPAP is discussed in this chapter because it shares many similar characteristics with other strategies of NPPV (e.g., lack of artificial airway, use of nasal or oronasal mask, use of airway pressures). In fact, when the inspiratory pressure and expiratory pressure of a bilevel positive airway pressure device are set at the same level, the effect is similar to that of CPAP.

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TABLE 7-2 Indication and Contraindications for Continuous Positive Airway Pressure

Indication	Contraindication
Obstructive sleep apnea	Apnea due to neuromuscular causes Progressive hypoventilation Fatigue of respiratory muscles Facial trauma Claustrophobia

✓ CPAP is the treatment of choice for obstructive sleep apnea without significant CO_2 retention.

CPAP is the treatment of choice for obstructive sleep apnea without significant carbon dioxide retention (Henderson & Strollo, 1999; Rosenthal, Nykamp & Guido et al., 1998). CPAP should be used with care and close monitoring of the patient as it is not effective in apnea due to neuromuscular causes. Table 7-2 outlines the indication and contraindications for CPAP therapy.

Obstructive Sleep Apnea

Sleep apnea is defined as a temporary cessation of breathing that lasts at least 10 sec during sleep (Wilkins & Dexter, 1998). Sleep apnea may be caused by airflow obstruction (obstructive sleep apnea), loss of neurologic breathing effort (central sleep apnea), or a combination of these two conditions (mixed sleep apnea). **Obstructive sleep apnea (OSA)** is diagnosed by nocturnal polysomnography and the severity is determined by the apnea and desaturation index (Arai et al., 1998; Redline & Strohl, 1998; Waite, 1998).

The severity of OSA is quantified by the **apnea index** (respiratory disturbance index). The apnea index represents the average number of apneas in one hour of sleep during a test. For example, if a patient slept for 8 hours during a test and had 160 apneas, the apnea index would be 20 (160/8). **Apnea-hypopnea index** refers to the average number of apnea or **hypopnea** in each hour of sleep during a test. **Desaturation index** describes the average number of oxygen desaturations of 4% or more from baseline (measured by pulse oximeter) in each hour of sleep during a test (Mooe et al., 2000).

Approximately 40 million Americans have chronic sleep disorders and the number of persons affected by OSA ranges from 2% to 4% of middle-aged adults. The distribution between genders is 4% to 9% in men and 1% to 2% in women. The incidence of OSA among morbidly obese patients is 12 to 30 times higher (Kyzer & Charuzi, 1998; Piccirillo et al., 1998; Skomro & Kryger, 1999).

Risk factors for OSA include history of snoring and witnessed apneas, obesity, increased neck circumference, hypertension, and family history of OSA (Skomro & Kryger, 1999). In patients with OSA, the major clinical signs and symptoms are snoring, daytime sleepiness, restless sleep, morning fatigue, and headaches. If untreated, OSA can lead to hypertension, left and right ventricular hypertrophy,

obstructive sleep apnea (OSA): OSA is caused by severe air flow obstruction during sleep.

apnea index: Average number of apneas in each hour of sleep during a test.

apnea-hypopnea index: Average number of apnea and hypopnea in each hour of sleep during a test.

hypopnea: Reduction in airflow for 10 or more seconds that is at least 50% below an estimated baseline amplitude, usually associated with oxygen desaturation or pulse alteration.

desaturation index: Average number of oxygen desaturations of 4% or more from baseline in each hour of sleep during a test.

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sudden cardiovascular death, and increased risk for brain infarction (Kyzer & Charuzi, 1998).

Treatments for OSA include oral applications such as prosthetic mandibular advancement (Ishida et al., 1998; Millman, Rosenberg & Kramer, 1998), surgical interventions such as tonsillectomy and uvulopalatopharyngoplasty for upper obstructions (Miyazaki, Itasaka & Tada et al., 1998; Powell, Riley & Robinson, 1998), and weight reduction gastric surgery for morbidly obese patients (Kyzer & Charuzi, 1998). Conservative therapies such as weight loss and patient positioning have been disappointing. CPAP has become the treatment of choice for the vast majority of patients with moderate to severe OSA (Henderson & Strollo, 1999; Rosenthal et al., 1998; Wilkins & Dexter, 1998). The procedure to titrate CPAP level is discussed later in this chapter.

USE OF BILEVEL POSITIVE AIRWAY PRESSURE (BILEVEL PAP)

bilevel positive airway pressure (bilevel PAP): Bilevel PAP has two pressure levels, whereas CPAP has only one.

positive end-expiratory pressure (PEEP): An airway pressure that is above 0 cm H_2O at end-expiration.

✓ Two indications for bilevel PAP are acute respiratory failure and acute hypercapnic exacerbations of COPD.

Bilevel positive airway pressure (bilevel PAP) differs from CPAP in that bilevel PAP has two pressure levels, whereas CPAP has only one. Bilevel PAP has an inspiratory positive airway pressure (IPAP) setting that provides mechanical breaths and an expiratory positive airway pressure (EPAP) level that functions as **positive end-expiratory pressure (PEEP)**. When bilevel PAP is used as an adjunct to provide mechanical ventilation, the two major indications are acute respiratory failure (Aboussouan, 2010; Abou-Shala, 1996; Jasmer, 1997; Keenan, 1997; Kramer et al., 1995; Wysocki et al., 1995) and acute hypercapnic exacerbations of COPD (Diaz et al., 1997; Girault et al., 1997). The most common criteria for the determination of acute respiratory failure are blood gas results. Typical results may show partially compensated respiratory acidosis with moderate hypoxemia (e.g., pH <7.35, $PaCO_2 > 50$ mm Hg, $PO_2 < 55$ mm Hg). For patients with hypoxemic respiratory failure, refractory hypoxemia may be present in addition to increasing PCO_2. A PaO_2/F_IO_2 (P/F) index of less than 250 mm Hg suggests presence of refractory hypoxemia (PaO_2 does not respond to high F_IO_2). In patients with acute cardiogenic pulmonary edema, CPAP or bilevel PAP ventilation has been found to reduce the need for subsequent mechanical ventilation (Peter et al., 2006).

Patients who are unable to use or tolerate a nasal or oronasal mask are not candidates for NPPV (e.g., facial trauma, claustrophobia, mouth breather, and lack of teeth). These patients may try a full-face mask. Inability to protect the airway from secretions or aspirations is also a contraindication for NPPV. NPPV should not be used in apneic patients. For apneic patients, traditional mechanical ventilation is indicated. Furthermore, patients who have acute respiratory distress should not be treated with NPPV, as this strategy may delay endotracheal intubation and initiation of mechanical ventilation. Delay to implement mechanical ventilation may lead to poor patient outcome (Wood et al., 1998). Table 7-3 outlines the common indications and contraindications for NPPV.

TABLE 7-3 Indications and Contraindications for Noninvasive Positive Pressure Ventilation

Indication	Contraindication
Reduction of respiratory workload in obesity	Apnea
Acute respiratory failure	Unable to handle secretions
Acute hypercapnic exacerbations of COPD	Facial trauma Claustrophobia

COMMON INTERFACES FOR CPAP AND BILEVEL PAP

✓ Nasal mask, oronasal mask, nasal pillows, and full-face mask are interfaces for NPPV.

Since CPAP or NPPV is done without an artificial airway, the interface between the patient and ventilator is typically an external device connecting the ventilator tubing to the patient's nose, mouth, or face. NPPV interfaces include nasal mask, oronasal mask, nasal pillows, and full-face mask.

Nasal Mask

nasal mask: A mask that covers only the nose.

The **nasal mask** is the most common interface used in NPPV for obstructive sleep apnea. A typical nasal mask has a soft cushion that surrounds and makes contact with the patient's nasal area. The soft cushion provides comfort to the patient while maintaining a seal to keep gas from leaking. While a tight seal is desirable, a minor leak is acceptable as it is not likely to compromise the effectiveness of NPPV. A common error is selecting a nasal mask that is too large for the patient. There are many nasal masks available. For patient comfort and compliance, it is a good practice to let the patient try out different sizes for maximal comfort and minimal leakage. Figure 7-1 shows the structure of a typical nasal mask (A) and when it is in use (B).

✓ A minor leak is acceptable when using a nasal mask. An oronasal mask should be considered when the leak is significant.

Since a nasal mask does not cover the mouth, leakage through the mouth is a common problem. This is particularly true when PPV is provided at high positive pressures. A minor leak is acceptable as long as ventilation and oxygenation are not compromised. An oronasal mask should be considered when the leak is significant or when ventilation is the primary indication for NPPV. During the initial setup of a nasal mask, pulse oximetry may be used to check for improvement in oxygenation and adequacy of oxygen saturation. Soon after stabilization of the patient, blood gases should be done to fine-tune settings on the ventilator so as to verify proper oxygenation, ventilation, and acid-base balance. Table 7-4 shows the advantages and disadvantages of the nasal mask.

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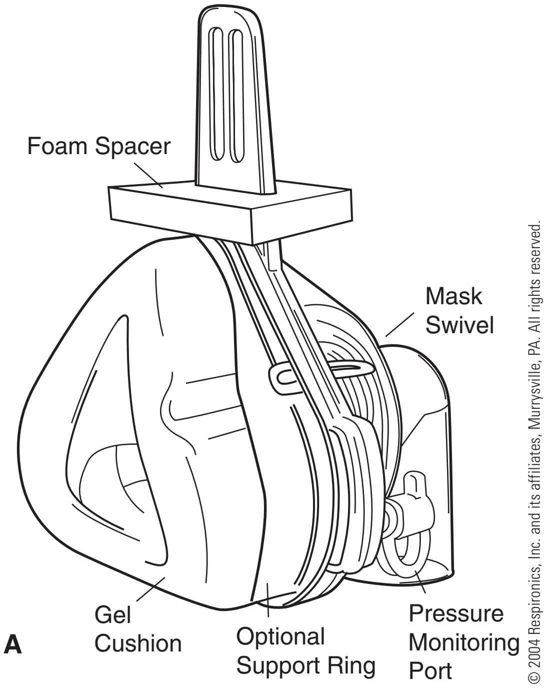
Foam Spacer
Mask
Swivel
Gel
Cushion
A
Optional
Support Ring
Pressure
Monitoring
Port

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FIGURE 7-1A Structure of a nasal mask.

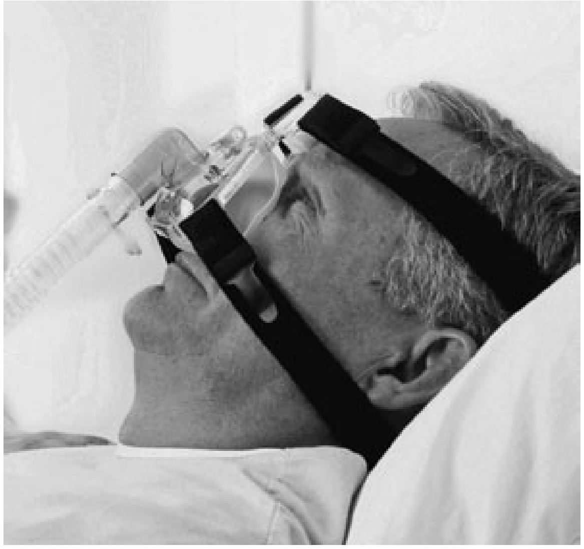

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FIGURE 7-1B Nasal mask in use.

TABLE 7-4 Advantages and Disadvantages of the Nasal Mask

Advantage	Disadvantage
Comfort	Gas leaks
Patient compliance	Nasal dryness or drainage

Oronasal Mask

oronasal mask: A mask that covers the nose and mouth. It is used in noninvasive positive pressure ventilation.

✓ Regurgitation and aspiration can be a potential problem when an oronasal mask is used.

✓ With an oronasal mask, asphyxiation can also be a problem in the event of ventilator failure, or electrical or gas source disconnection.

The function of an **oronasal mask** (Figure 7-2) is essentially the same as a nasal mask. It has a soft cushion that surrounds and makes contact with the patient's nasal and oral area. As does the nasal mask, the soft cushion also provides comfort to the patient while maintaining a seal to keep gas from leaking. With an oronasal mask, a tight seal is easier to maintain than with a nasal mask because both the nose and mouth are covered. However, because the mouth is covered, regurgitation and aspiration can be potential problems with the use of an oronasal mask. Asphyxiation can also occur in the event of ventilator failure, or electrical or gas source disconnection. Anti-asphyxia valves provide a safety opening in these situations. Proper disconnection alarms and patient monitors must be functional during the use of NPPV via an oronasal mask. Table 7-5 shows the advantages and disadvantages of the oronasal mask.

Some patients may benefit from a total face mask which covers the eyes, nose, and mouth. Total head interface is also available for patients who may have trouble with leaks or proper fitting of other interfaces.

Nasal Pillows

nasal pillows: Nasal pillows are a small interface commonly used for CPAP therapy.

Nasal pillows (nasal prongs) is another interface that resembles a nasal mask but is smaller. Nasal pillows devices are more commonly used during CPAP therapy. This interface consists of two small cushions that fit snugly within the nostrils. Because nasal pillows do not come into contact with the lips, chin or

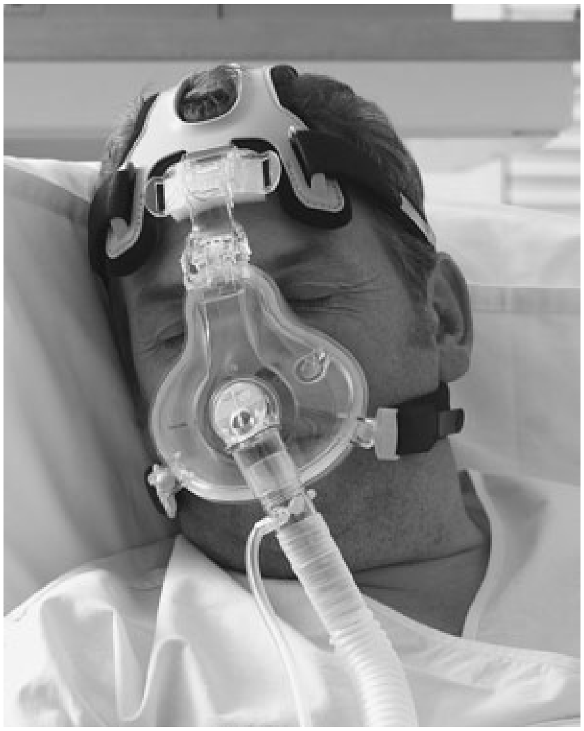

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FIGURE 7-2 An oronasal mask in use.

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TABLE 7-5 Advantages and Disadvantages of the Oronasal Mask

Advantage	Disadvantage
Good seal	Claustrophobia
More effective ventilation	Patient noncompliance Regurgitation and aspiration Asphyxiation in power or gas outage Alarm and monitor are necessary

cheeks, they are an alternative to nasal, oronasal, or full-face masks. Since this interface has a lower pressure range (e.g., 3 to 20 cm H_2O), nasal pillows devices are not as effective as the larger nasal or oronasal mask when used in the bilevel PAP mode. Some common complaints of nasal pillows are nasal congestion, nose bleed, and dry or sore mouth. A humidifier or saline nasal spray may help to minimize these side effects. Figure 7-3 shows the structure of nasal pillows and Figure 7-4 shows the insertion, placement, and adjustment of this interface.

Nasal pillows devices are more comfortable than oronasal masks, but gas leak is a potential problem especially in men with beards and mustaches. If significant gas leaks occur through the mouth, a chin strap may be used to close the mouth or an oronasal mask should be considered.

✓ A chin strap to close the mouth or an oronasal mask should be considered when gas leak through the mouth is significant.

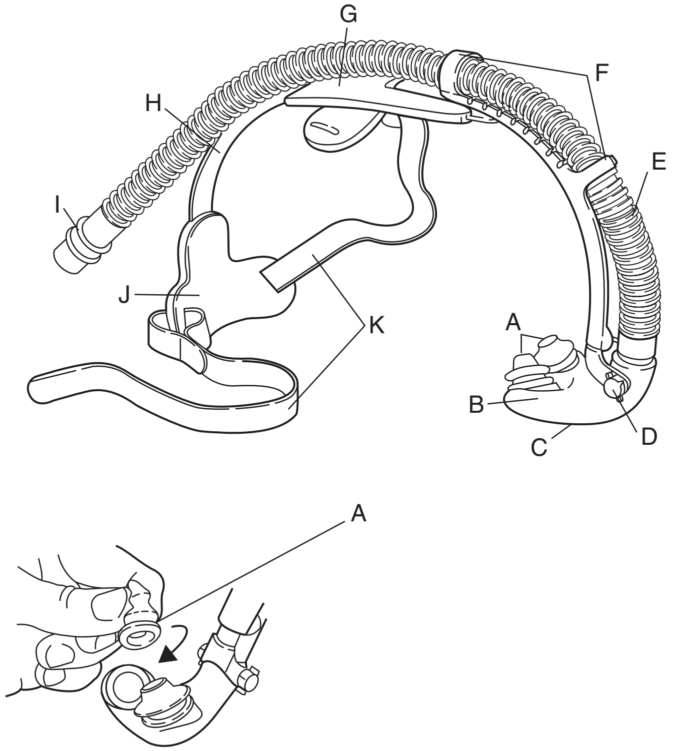

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FIGURE 7-3 Structure of a nasal pillows interface. (A) Nasal Pillows™; (B) Plenum shell; (C) Vent-hole insert; (D) Wing knob (to adjust the angle of plenum shell); (E) Hose; (F) Hose guide; (G) Outrigger; (H) Spine; (I) Swivel adapter; (J) Cradle; (K) Side straps.

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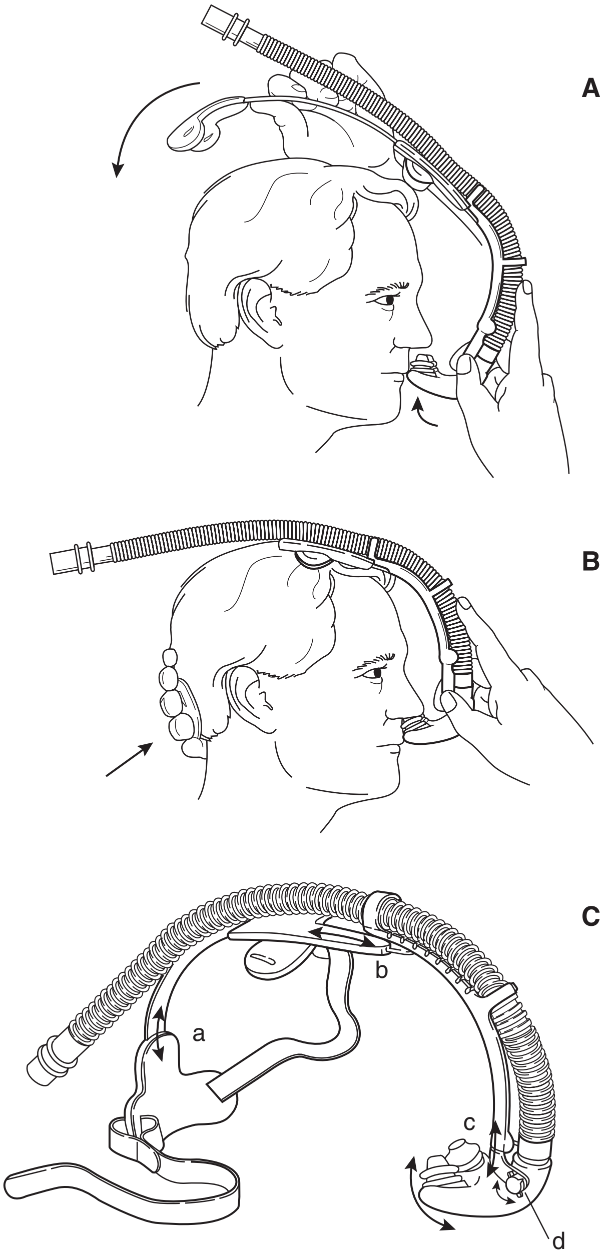

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FIGURE 7-4 Use of a nasal pillows interface. (A) Insert the Nasal Pillows™ into nostrils; (B) Place the cradle (back pad) below the curve at the back of head; (C) For a comfortable fit, adjust the following points: (a) height of the cradle; (b) length of unit; (c) height of plenum shell; (d) angle of plenum shell.

Full-Face Mask

full-face mask: An interface that covers almost the entire face of a patient.

The **full-face mask** covers almost the entire face of a patient. It is easy to fit and apply for new patients because of its large size. The size of this mask is able to cover the nose bridge and mouth without making direct contacts. For this reason, it

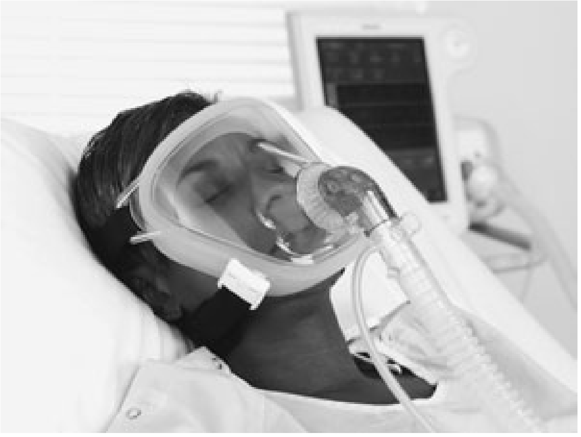
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FIGURE 7-5 A full-face mask in use.

does not inherit problems with air leaks and pressure sores around the nose and mouth. The full-face mask is ideal for claustrophobic patients, mouth breathers, and patients without teeth or with facial abnormalities (Philips, 2012). Figure 7-5 shows a full-face mask.

POTENTIAL PROBLEMS WITH INTERFACES

Since patients are different in size and facial structure, the selection of an interface must be done on an individual basis. Following selection of an interface, a trial period usually is required for the patient to get used to the interface and to identify problems with it.

TITRATION OF CONTINUOUS POSITIVE AIRWAY PRESSURE

✓ The initial CPAP is started at 4 cm H_2O, and titrated to a desired endpoint.

When positive airway pressure is used to treat or relieve OSA, the appropriate CPAP level is done by setting the CPAP level or by adjusting the inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) at the same level, initially at 4 cm H_2O. After setting up the machine and placing the interface on the patient, pulse oximetry readings and number of apnea episodes during polysomnography are used to fine-tune the CPAP level.

Autotitration

Autotitration (autoadjusting) CPAP equipment are available for long-term home treatment to improve effectiveness by ensuring that the pressure is near optimal to the patient's needs. The working mechanism by which these equipment titrate

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pressures varies greatly. Some equipment detects snoring while others detect inspiratory flow patterns or airway vibration. In patients with uncomplicated OSA, unattended autotitration of CPAP may be suitable for the relief of airway obstruction. Autotitration should be used following thorough medical screening and complete patient education on the use of the prescribed CPAP device (Berthon-Jones et al., 1996; Teschler et al., 1996).

Unattended autotitration should not be used in patients with central sleep apnea, cerebral vascular accident, prolonged hypoventilation, hypovolemia, or cardiac failure. Other potential contraindications to autotitrating CPAP equipment include uncontrolled asthma, epilepsy, angina, fluid and electrolyte disturbances, nocturnal myoclonus, and other parasomnias. Severe air leak around the mask should be corrected before implementing autotitrating CPAP (Teschler et al., 1998).

Ramp

Ramp is a feature in which the starting pressure (i.e., preset or adjustable) gradually increases over time (e.g., up to 45 min) until the desired pressure is reached. This feature is ideal for patients who may have trouble tolerating a sudden onset of high pressure.

C-Flex™

C-Flex™ is a method of delivering CPAP for the treatment of OSA. A common complaint by patients is that they have trouble exhaling against the continuous positive pressure. This problem may result in frequent awakening during sleep, sleep fragmentation, and occasional noncompliance in the use of the equipment. C-Flex™ provides pressure relief during exhalation. This reduces the continuous pressure which the patient must overcome during exhalation.

C-Flex™ is titrated in the same way as conventional CPAP. During use, the equipment monitors the airflow during exhalation and reduces expiratory pressure proportional to expiratory flow. The relief pressure is provided on a breath-to-breath basis, depending on the actual expiratory airflow. Prior to the end of expiration and start of inspiration, the preset CPAP level is restored. Three levels of C-Flex™ (1, 2, 3) are available and each higher level provides progressively increased pressure relief.

TITRATION OF BILEVEL POSITIVE AIRWAY PRESSURES

A typical starting point for BiPAP™ is 8/4 (8 cm H_2O inspiratory and 4 cm H_2O expiratory) and titrated to an appropriate level.

When positive airway pressure is used to support acute respiratory failure, the IPAP and EPAP levels are set independently. Bilevel refers to the inspiratory and expiratory positive airway pressure settings. For example, a bilevel positive airway pressure setting of 8 and 4 means that the IPAP is 8 cm H_2O and the EPAP is 4 cm H_2O. Common *initial* pressure settings are IPAP from 8 to 12 cm H_2O and EPAP from 4 to 5 cm H_2O (Hill, 2004). The procedure for titration of IPAP and EPAP (ResMed Corp., 1998a) is outlined in Table 7-6.

TABLE 7-6 Procedure to Titrate Bilevel Positive Airway Pressure

1. Set mode: spontaneous/timed.
2. Start the IPAP at 8 cm H_2O and the EPAP at 4 cm H_2O.
3. Set IPAP maximum time 0.15 to 0.25 sec longer than the patient's actual inspiratory time. IPAP maximum time should not be set longer than 50% of the respiratory cycle.
4. Attach mask to patient, ensure proper fit, and start machine.
5. Increase IPAP in 1 to 2 cm H_2O increments to provide more ventilatory assistance and larger tidal volume.
6. Increase EPAP in 1 to 2 cm H_2O increments to improve oxygenation or to relieve upper airway obstruction.
7. If poor synchronization occurs, check for leaks or alter IPAP maximum time to improve synchronization.
8. Use supplemental oxygen if baseline saturation remains low with appropriate IPAP and EPAP settings.
9. Do not increase IPAP or EPAP level beyond patient tolerance.

Bi-Flex™

Bi-Flex™ is a method of delivering bilevel PAP in which the airflow during inhalation and exhalation is "softened." This makes breathing more natural and comfortable for the patient. This mode provides pressure relief at the end of inhalation and the start of exhalation (Figure 7-6). Three levels of patient-adjustable Bi-Flex™ (1, 2, 3) are available and each higher level provides progressively increased pressure relief.

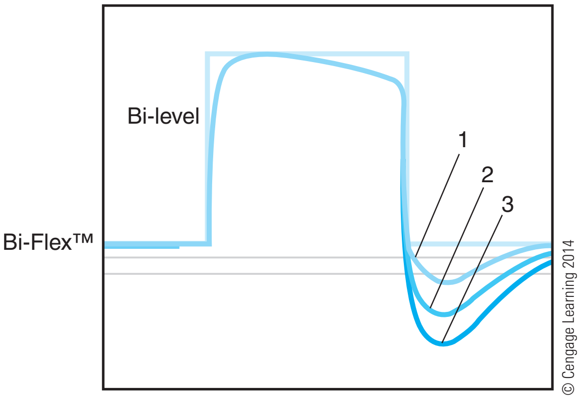

FIGURE 7-6 Bi-Flex™ provides pressure relief at the end of inhalation and the start of exhalation. Level 3 provides the highest level of pressure relief as indicated by the pressure drop below the baseline pressure.

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SUMMARY

Noninvasive strategies of assisted breathing have been used successfully in providing sufficient ventilation and oxygenation support to patients with obstructive sleep apnea (CPAP), acute ventilatory failure, and impending ventilatory failure (bilevel positive airway pressure). The two advantages of NPPV are (1) an artificial airway is not necessary and (2) lower risks associated with endotracheal intubation and traditional mechanical ventilation.

As with other noninvasive procedures applied to a potentially critical condition, close monitoring of the patient is an absolute requirement. Use of accessory muscles, changes in vital signs, and signs of respiratory distress or hypoxia are some indications that the patient's condition is deteriorating. The patient must be assessed in a timely manner. If indicated, the patient should be intubated and mechanically ventilated if NPPV fails to stabilize or improve the patient's condition.

Self-Assessment Questions

1. Which of the following is *not* true in regard to noninvasive positive pressure ventilation (NPPV)?

   A. NPPV requires the use of an artificial airway.
   B. NPPV can provide positive end-expiratory pressure (PEEP).
   C. NPPV may be used to assist patients with obstructive sleep apnea.
   D. NPPV may be used to assist patients with acute ventilatory failure.

2. The titration endpoints of IPAP and EPAP during bilevel PAP may include all of the following measurements *except*:

   A. SpO_2.
   B. PaO_2.
   C. PvO_2.
   D. $P_{ET}CO_2$.

3. Continuous positive airway pressure (CPAP) provides positive airway pressure during ________ and it functions as ________.

   A. spontaneous breathing, NPPV
   B. spontaneous breathing, PEEP
   C. mechanical ventilation, NPPV
   D. mechanical ventilation, PEEP

4. When CPAP is in use, the total amount of work of breathing is provided by the:

   A. patient.
   B. ventilator.
   C. patient and ventilator.
   D. pressure level of CPAP.

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5. CPAP is the treatment of choice for _______ sleep apnea without significant _______.

   A. central, hypoxemia
   B. central, carbon dioxide retention
   C. obstructive, hypoxemia
   D. obstructive, carbon dioxide retention

6. All of the following clinical conditions are contraindications for CPAP *except*:

   A. acute respiratory acidosis.
   B. impending ventilatory failure.
   C. obesity.
   D. neuromuscular disease.

7. Sleep apnea is defined as a temporary cessation of breathing that lasts at least _______ during sleep.

   A. 5 sec
   B. 10 sec
   C. 30 sec
   D. 60 sec

8. During NPPV, mechanical breaths are provided by the _______ setting, whereas the end-expiratory pressure is determined by the _______ setting.

   A. IPAP, EPAP
   B. EPAP, IPAP
   C. PEEP, IPAP
   D. PEEP, EPAP

9. The interfaces for NPPV include all of the following devices *except*:

   A. nasal mask.
   B. endotracheal tube.
   C. nasal pillows.
   D. oronasal mask.

10. Which of the following interfaces is *least* likely to develop air leaks and pressure sores?

    A. nasal mask
    B. oronasal mask
    C. nasal pillows
    D. full-face mask

11. The oronasal mask is a NPPV interface that has all of the following inherited risks *except*:

    A. aspiration.
    B. asphyxiation.
    C. regurgitation.
    D. hyperventilation.

12. The initial CPAP level is started at _______ cm H_2O and titrated to a desired endpoint.

    A. 2
    B. 4
    C. 6
    D. 8

13. The typical starting point for bilevel positive airway pressure is started at _______ cm H_2O (inspiratory) and _______ cm H_2O (expiratory) and subsequently titrated to a desired level.

    A. 2, 6
    B. 4, 8
    C. 6, 2
    D. 8, 4

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14. During titration of bilevel positive airway pressure, the _______ is increased in 1 to 2 cm H_2O increments to provide more ventilatory assistance and larger tidal volume.

A. PEEP
B. CPAP
C. IPAP
D. EPAP

15. During titration of bilevel positive airway pressure, the _______ is increased in 1 to 2 cm H_2O increments to improve oxygenation or to relieve upper airway obstruction.

A. IPAP
B. EPAP
C. peak inspiratory pressure
D. plateau pressure

16. Ramp is a strategy used in CPAP to allow the patient to accept a gradual:

A. increase in inspiratory pressure.
B. increase in inspiratory flow.
C. decrease in inspiratory pressure.
D. decrease in inspiratory flow.

Answers to Self-Assessment Questions

1. A.
2. C.
3. B.
4. A.
5. D.
6. C.
7. B.
8. A.
9. B.
10. D.
11. D.
12. B.
13. D.
14. C.
15. B.
16. A.

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Chapter 8

Initiation of Mechanical Ventilation

David W. Chang
James H. Hiers

Outline

- Introduction
- Goals of Mechanical Ventilation
- Indications
  - *Acute Ventilatory Failure*
  - *Impending Ventilatory Failure*
  - *Severe Hypoxemia*
  - *Prophylactic Ventilatory Support*
- Contraindications
- Initial Ventilator Settings
  - *Mode*
  - *Dual Control Mode*
  - *Frequency*
  - *Tidal Volume*
  - *Pressure Support*
  - *F_IO_2*
  - *PEEP*
  - *I:E Ratio*
  - *Flow Pattern*
- Ventilator Alarm Settings
  - *Low Exhaled Volume Alarm*
  - *Low Inspiratory Pressure Alarm*
  - *High Inspiratory Pressure Alarm*
  - *Apnea Alarm*
  - *High Frequency Alarm*
  - *High and Low F_IO_2 Alarms*
- Hazards and Complications
  - *Types of Hazards and Complications*
  - *Malfunction and Misuse of Alarms*
  - *Barotrauma*
  - *Decrease in Cardiac Output and Blood Pressure*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

acute ventilatory failure

alveolar-arterial oxygen pressure gradient [$P_{(A-a)}O_2$]

circuit compressible volume

dual control mode

flow rate

I:E ratio
impending ventilatory failure
maximum inspiratory pressure (MIP)
medical futility
P/F ratio
prophylactic ventilatory support

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Outline the goals of mechanical ventilation.
- Provide clinical signs and examples to differentiate the four primary indications for mechanical ventilation.
- List the contraindications for mechanical ventilation.
- Provide the suggested initial ventilator settings for mode, frequency, tidal volume, pressure support, F_IO_2, PEEP, I:E ratio, and flow pattern.
- Provide the suggested initial ventilator alarm settings for low volume, high/low pressures, apnea, high frequency, and high/low F_IO_2.
- Name the hazards and complications of mechanical ventilation.

INTRODUCTION

This chapter covers the indications, contraindications, and strategies for the initiation of mechanical ventilation. Once a decision is made to implement mechanical ventilation, the initial settings on the ventilator must be made using a systematic approach. It is important to remember that these initial settings are subject to change according to the changing patient condition. Frequent monitoring of the patient is necessary to use the ventilator properly and to improve the patient outcome.

GOALS OF MECHANICAL VENTILATION

Mechanical ventilation alone does not treat or reverse the underlying pathology leading to the need for ventilator support. Rather, it is applied as one of the support systems until the reversal of the pathological condition, so that the patient may then become weaned from mechanical ventilation. Table 8-1 outlines the five main goals of mechanical ventilation (Tobin, 1994).

TABLE 8-1 Goals of Mechanical Ventilation

Goal	Target
1. Improve gas exchange	Reverse hypoxemia Relieve acute respiratory acidosis
2. Relieve respiratory distress	Reduce oxygen cost of breathing Reverse respiratory muscle fatigue
3. Improve pulmonary mechanics	Prevent and reverse atelectasis Improve compliance Prevent lung injury
4. Permit lung and airway healing	Maintain lung and airway functions
5. Avoid complications	Protect lung and airway Prevent disuse respiratory muscle dystrophy

INDICATIONS

✓ Mechanical ventilation is indicated when the patient cannot maintain spontaneous ventilation to provide adequate oxygenation or carbon dioxide removal.

Mechanical ventilation is indicated when the patient cannot maintain spontaneous ventilation to provide adequate oxygenation or carbon dioxide removal. The clinical conditions leading to mechanical ventilation can be grouped into four areas: (1) **acute ventilatory failure**; (2) **impending ventilatory failure**; (3) severe hypoxemia; and (4) **prophylactic ventilatory support** (Brown, 1994; Otto, 1986). Table 8-2 outlines the indications for mechanical ventilation.

acute ventilatory failure: An increase of $PaCO_2$ (>50 mm Hg) with a concurrent decrease of arterial (pH <7.30).

impending ventilatory failure: A gradual increase of $PaCO_2$ (>50 mm Hg) caused by deteriorating lung functions.

prophylactic ventilatory support: Early intervention of potential ventilatory failure by means of mechanical ventilation.

Acute Ventilatory Failure

The primary indication for mechanical ventilation is acute ventilatory failure. This is defined as a sudden increase in the $PaCO_2$ to greater than 50 mm Hg with an accompanying respiratory acidosis (pH <7.30). In the COPD patient, mechanical ventilatory support is indicated by an acute increase in the $PaCO_2$ above the patient's normal baseline $PaCO_2$ accompanied by a decompensating respiratory acidosis (Brown, 1994; Otto, 1986).

Other signs that may be useful in the assessment of acute ventilatory failure include apnea, bradypnea, ALI, and ARDS (Byrd et al., 2010). However, mild to moderate hypoxemia (PaO_2 = 50 to 60 mm Hg or SaO_2 = 85% to 90%) does not by itself indicate the presence of acute ventilatory failure or the need for ventilatory support. If a hypoxemic patient is able to maintain adequate ventilation as

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TABLE 8-2 Indications for Mechanical Ventilation

Indication	Examples
1. Acute ventilatory failure	Apnea or bradypnea Acute lung injury (ALI) or Acute respiratory distress syndrome (ARDS) pH <7.30, $PaCO_2$ >50 mm Hg
2. Impending ventilatory failure	Progressive acidosis and hypoventilation to pH <7.30 and $PaCO_2$ >50 mm Hg Spontaneous frequency >30/min
3. Severe hypoxemia	PaO_2 <40 mm Hg, SaO_2 <75% PaO_2/F_IO_2 (**P/F ratio**): ≤300 mm Hg for ALI, ≤200 mm Hg for ARDS
4. Prophylactic ventilatory support	Postanesthesia recovery Muscle fatigue Neuromuscular disease

P/F ratio: PaO_2/F_IO_2. Clinical assessment of oxygenation and degree of hypoxemia, ≤300 mm Hg for ALI, ≤200 mm Hg for ARDS.

TABLE 8-3 Assessment of Acute Ventilatory Failure

Parameter	Limit
$PaCO_2$	>50 mm Hg (higher for COPD patients)
pH	<7.30

documented by the $PaCO_2$, then the patient may be supported with supplemental oxygen or continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP). Table 8-3 shows some common methods to assess the presence of acute ventilatory failure.

Impending Ventilatory Failure

Impending ventilatory failure occurs when a patient can maintain only marginally normal blood gases, but only at the expense of a significantly increased work of breathing. Depending on the pulmonary reserve and lung function of a patient, the $PaCO_2$ value may be normal or low at the beginning of impending ventilatory failure. This is because of an increase in minute ventilation in an attempt to

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compensate for the gas exchange deficiencies. However, if the underlying pathology is not corrected in time, ventilatory failure will ensue when muscle fatigue occurs as a result of prolonged, excessive work of breathing. At this time, the $PaCO_2$ will rise and the pH will fall.

If the early clinical signs indicate that a patient is in impending ventilatory failure, it is appropriate to initiate mechanical ventilation. Early intervention is done to correct hypoxemia and acidosis imposed on the major organs and to reduce the stress placed on the cardiopulmonary system. There are several objective measurements that can be used to determine whether the patient is in impending ventilatory failure. These measurements are discussed below.

✔ Assessment of impending ventilatory failure relies on the measurements related to the lung functions (i.e., V_T, f, V_E, VC, MIP).

Assessment of Impending Ventilatory Failure. Development of impending ventilatory failure is dependent on the balance of metabolic needs and work of breathing. When metabolic needs cause excessive work of breathing, impending ventilatory failure is likely. Since the work of breathing is carried out entirely by the respiratory system, assessment of impending ventilatory failure relies solely on the measurements related to the lung functions (i.e., tidal volume, frequency, minute volume, vital capacity, and maximal inspiratory pressure). Table 8-4 shows the factors for the assessment of impending ventilatory failure.

Tidal volume. A spontaneous tidal volume of less than 3 to 5 mL/kg is indicative of impending ventilatory failure.

Frequency (f). A spontaneous frequency of greater than 30/min may indicate impending ventilatory failure (Byrd et al., 2010).

Minute volume. If the patient's spontaneous minute volume is greater than 10 L/min, then impending ventilatory failure is likely. Although it may appear that

TABLE 8-4 Assessment of Impending Ventilatory Failure

Parameter	Threshold
Tidal volume	<3 to 5 mL/kg
Frequency and pattern	>30/min Labored or irregular respiratory pattern
Minute ventilation	>10 L/min
Vital capacity	<15 mL/kg
Maximum inspiratory pressure (MIP)	<-20 cm H_2O
$PaCO_2$ trend	Increasing to over 50 mm Hg
Vital signs	Increase in heart rate, blood pressure

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an increasing minute volume is a sign of improving lung functions, in actuality the patient may not be able to sustain the increased work of breathing. Muscle fatigue can occur over time and lead to eventual ventilatory failure. In addition, an increase in minute volume achieved by an increased frequency and a decreased tidal volume lead to a larger percentage of deadspace (wasted) ventilation. This condition increases the oxygen cost of breathing and carbon dioxide production, progressive hypercapnia, and hypoxemia.

Vital capacity. If the patient's vital capacity is less than 15 mL/kg, then impending ventilatory failure is likely. An accurate measurement of vital capacity requires patient cooperation, which may be difficult to achieve during impending ventilatory failure. The maximum inspiratory pressure measurement can be used as its alternative if the patient is unable to perform the vital capacity maneuver.

maximum inspiratory pressure (MIP): Also called negative inspiratory force (NIF). MIP reflects a patient's respiratory muscle strength. MIP of less than –20 cm H_2O (e.g., –10 cm H_2O) is one of the indications for impending ventilatory failure. It is obtained by measuring the maximum negative pressure during a forced inspiratory maneuver against a closed manometer.

Maximum inspiratory pressure. The **maximum inspiratory pressure (MIP)** is a measure of the inspiratory muscle strength reflecting the patient's pulmonary reserves. Patients with an MIP of greater than −25 cm H_2O obtained within 20 sec can be assumed to have a vital capacity of 15 mL/kg (Shapiro et al., 1991). When MIP is less than −20 cm H_2O, it is one of the signs of impending ventilatory failure.

The MIP is obtained by measuring the maximum negative pressure that the patient can generate with a forced inspiratory maneuver against a negative manometer (pressure measuring device). Although the MIP maneuver can be performed using a face mask, it is easier to obtain with an endotracheal or tracheostomy tube.

MIP can be measured by using a T-piece with one port attached to the endotracheal or tracheostomy tube, one port attached to the negative pressure manometer, and one port attached to a special unidirectional valve that allows exhalation only. The patient is encouraged to exhale to residual volume and then inhale as forcefully as possible. The unidirectional valve allows the patient to exhale so that the subsequent MIP maneuvers can be performed from the residual volume level (Caruso et al., 1999).

$PaCO_2$ trend. A gradual but persistent increase of the $PaCO_2$ to more than 50 mm Hg is indicative of impending ventilatory failure. The $PaCO_2$ measurements should be done over a period of time and on an as-needed basis. The $PaCO_2$ should be interpreted along with the patient's breathing pattern since progressive tachypnea is common during impending ventilatory failure.

Vital signs. Any clinical indicators that show a patient is under distress or is tiring must also be considered when assessing for presence of impending ventilatory failure. These indicators include tachycardia, arrhythmias, hypertension, tachypnea, use of accessory respiratory muscles, diaphoresis, and cyanosis.

✓ Severe hypoxemia is present when the PaO_2 is less than 60 mm Hg on 50% or more of oxygen or less than 40 mm Hg at any F_IO_2.

Severe Hypoxemia

Hypoxemia is a common finding in lung diseases. When hypoxemia is severe, mechanical ventilation may be necessary to support the oxygenation deficit. ALI,

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ARDS, pulmonary edema, and carbon monoxide poisoning are examples that often require ventilatory support for the primary purpose of oxygenation.

alveolar-arterial oxygen pressure gradient [$P_{(A\text{-}a)}O_2$]: The difference of P_AO_2 and PaO_2. A gradient over 450 mm Hg while on 100% oxygen indicates severe hypoxemia or intrapulmonary shunting.

See Appendix 1 for example.

Hypoxemia can be assessed by measuring the PaO_2, or the **alveolar-arterial oxygen pressure gradient [$P_{(A\text{-}a)}O_2$]**. Severe hypoxemia is present when the PaO_2 is less than 60 mm Hg on 50% or more of oxygen or less than 40 mm Hg at any F_IO_2. $P_{(A\text{-}a)}O_2$ is the difference of P_AO_2 and PaO_2. The normal $P_{(A\text{-}a)}O_2$ at 21% F_IO_2 should be less than 4 mm Hg for every 10 years of age. On 100% oxygen, every 50-mm Hg difference in $P_{(A\text{-}a)}O_2$ approximates 2% shunt (Shapiro et al., 1994).

$$P_{(A\text{-}a)}O_2 = P_AO_2 - PaO_2$$

PaO_2 is obtained from arterial blood gas analysis and P_AO_2 can be calculated as follows:

A simplified alveolar air equation:

$$P_AO_2 = (P_B - PH_2O) \times F_IO_2 - (PaCO_2/R)$$

$PaCO_2/0.8$ may be changed to $PaCO_2 \times 1.25$.

where P_B = barometric pressure, PH_2O = water vapor pressure (47 mm Hg at 37°C), and R = respiratory quotient (estimated to be 0.8). P_AO_2 is mainly affected by changes of F_IO_2, $PaCO_2$, and P_B.

PCWP of ≤18 mm Hg is used to rule out pulmonary edema or bilateral infiltrates caused by *cardiogenic* pulmonary edema.

Patients with ALI and ARDS share three common clinical manifestations: acute onset, bilateral infiltrates on frontal chest radiograph, and normal pulmonary capillary wedge pressure (PCWP) of ≤18 mm Hg. The only distinguishing feature separating ALI and ARDS is the degree of hypoxemia or PaO_2/F_IO_2 (P/F) ratio. The threshold for ALI is a P/F value ≤300 mm Hg. For ARDS, the P/F threshold is ≤200 mm Hg (Bernard et al., 1994). Since severe hypoxemia is the hallmark of ALI and ARDS, the P/F ratio can be used to assess the degree of hypoxemia in critically ill patients. The P/F ratio is calculated by:

$$P/F = (PaO_2 \,/\, F_IO_2) \text{ mm Hg}$$

Prophylactic Ventilatory Support

Prophylactic ventilatory support is provided in clinical conditions in which the risk of pulmonary complications, ventilatory failure, or oxygenation failure is high.

Prophylactic ventilatory support is provided in clinical conditions in which the risk of pulmonary complications, ventilatory failure, or oxygenation failure is high. In addition, prophylactic or early commitment of the patient to the ventilator can minimize hypoxia of the major body organs. It can also reduce the work of breathing and oxygen consumption and thus preserve and rest the cardiopulmonary system, and promote patient recovery (Otto, 1986). Indications for prophylactic ventilatory support are outlined in Table 8-5.

Untreated tension pneumothoraxis is an absolute contraindication for mechanical ventilation.

CONTRAINDICATIONS

Since positive pressure ventilation is contraindicated in untreated tension pneumothorax, mechanical ventilation at any positive pressure level must not be done without a functional chest tube to relieve the pleural pressure. Other contraindications

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TABLE 8-5 Indications for Prophylactic Ventilatory Support

Indication	Examples
Reduce risk of pulmonary complications	Prolonged shock Head injury Smoke inhalation
Reduce hypoxia of major body organs	Hypoxic brain Hypoxia of heart muscles
Reduce cardiopulmonary stress	Prolonged shock Coronary artery bypass surgery Other thoracic or abdominal surgeries

for mechanical ventilation are relative in nature and deal with the condition and prognosis of the patient.

There are three considerations in which mechanical ventilation should be terminated or should not be started (Campbell et al., 1992). They are based on (1) patient's informed request, (2) **medical futility**, and (3) reduction or termination of patient pain and suffering.

medical futility: A condition in which medical interventions are useless based on past experience.

A patient's informed request carries with it many legal, ethical, medical, and economical concerns. The health care facilities and professionals should be prepared to work with the patient and his family on this type of request. A protocol should be established to serve as a guide before a hasty decision is needed concerning whether ventilatory support should be started or discontinued.

Schneiderman et al. (1990) suggested that medical intervention may be futile if the physicians have concluded that intervention was useless in the last 100 similar cases. It is reasonable to infer that if medical intervention will not be effective in all probability, life support measures including mechanical ventilation should not be started. In this case, the physician must establish an open, honest discussion with the patient and the concerned parties about the potential outcomes for withholding ventilatory support.

Another relative contraindication for mechanical ventilation is to reduce or terminate patient pain and suffering. In probable terminal cases such as metastatic cancer and multiorgan failure, the benefit of mechanical ventilation must be weighed against the degree of pain and expected length of suffering that a patient may be subjected to. Physical restraints, painful and uncomfortable medical procedures, and psychological trauma are just a few problems that may not be completely alleviated by sedatives and analgesics (Campbell et al., 1992). Of course, medical futility would be a concurrent concern in dealing with this question.

Withdrawing ventilatory support from a patient poses a greater challenge than withholding mechanical ventilation. But this difficult decision should be based on the fact that mechanical ventilation is a supportive measure, rather than a curative procedure.

INITIAL VENTILATOR SETTINGS

When it becomes necessary to provide mechanical ventilatory support for a patient, the following basic ventilator settings must be determined: mode, frequency, tidal volume, F_IO_2, inspiratory:expiratory ratio, inspiratory flow pattern, and various alarm limits.

These initial ventilator settings are mainly based on a patient's body size, diagnosis, pathophysiology, and laboratory results. These settings only serve as a starting point and they should be adjusted according to changes in the patient's condition and requirements.

Mode

✓ Full ventilatory support may be necesssary if the patient is not breathing spontaneously between mechanical breaths.

✓ Partial ventilatory support is achieved by any mode that provides less than the total amount of the work of breathing.

The first step in selecting the ventilator mode is to decide whether the patient should receive full ventilatory support (FVS) or partial ventilatory support (PVS). Full ventilatory support is achieved by any mode that assumes essentially all of the work of breathing. The majority of ventilator patients initially require full support, with the control mode or the assist/control mode. The synchronized intermittent mandatory ventilation (SIMV) mode also provides full ventilatory support if the patient is not breathing spontaneously between mechanical breaths, and the mandatory frequency is set at 12/min or higher.

Partial ventilatory support is achieved by any mode that provides less than the total amount of the work of breathing. Partial support would be inappropriate initially for patients with ventilatory failure, and it is more commonly used during the weaning process. Some examples are bilevel positive airway pressure (BiPAP), and pressure support ventilation (PSV). These topics are discussed elsewhere in this text.

Dual Control Mode

dual control mode: A combined mode between two control variables (e.g., pressure and volume) that are regulated by independent feedback loops so that the delivered breath switches between pressure-controlled and volume-controlled.

In traditional ventilation, a single control variable is selected to achieve a desired goal. Examples of the single-variable control include volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV). In VCV, a desired tidal volume (a control variable) is set and the pressure changes from breath to breath depending on the characteristics of the patient-ventilator system. In PCV, the desired pressure (a control variable) is set and the delivered volume changes according to the characteristics of the patient-ventilator system (Campbell et al., 2002).

A **dual control mode** combines two control variables (e.g., pressure and volume) that are regulated by independent feedback loops so that the delivered breath switches between pressure-controlled and volume-controlled. In short, a dual control mode is a combined mode between two control variables. When VCV and PCV are combined, the patient receives mandatory breaths that are volume-targeted, pressure-limited, and time-cycled (Campbell et al., 2002). Since there are many dual control modes on the market and more are forthcoming in the future, selection

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of any mode of ventilation (single or dual) must be based on sound scientific basis and controlled clinical trials. Furthermore, the patient must be monitored and evaluated on a regular frequency in order to ensure the safety and effectiveness of the mode of ventilation.

Frequency

The initial ventilator frequency should be set between 10 and 12/min.

High ventilator frequency, inadequate inspiratory flow, and air trapping contribute to the development of *auto-PEEP*.

The initial ventilator frequency is the number of breaths per minute that is intended to provide eucapneic ventilation ($PaCO_2$ at patient's normal). The initial frequency is usually set between 10 and 12/min. This frequency, coupled with a 10 to 12 mL/kg tidal volume, usually produces a minute volume that is sufficient to normalize the patient's $PaCO_2$. Frequencies of 20/min or higher are associated with auto-PEEP and should be avoided (Shapiro, 1994). In addition to high ventilator frequency, inadequate inspiratory flow and air trapping contribute to the development of *auto-PEEP*.

An alternative method of selecting the initial frequency is to estimate the patient's minute volume requirement and divide the estimated minute volume by the tidal volume.

$$\text{Frequency} = \frac{\text{Estimated minute volume}}{\text{Tidal volume}}$$

The estimated minute volume for males is equal to 4.0 multiplied by the body surface area (BSA) and for females is equal to 3.5 multiplied by the BSA. The BSA (in square meters) can be obtained from a nomogram such as the Dubois body surface area chart (Appendix 2).

$$\text{Minute Volume (Male)} = (4)(\text{BSA})$$

$$\text{Minute Volume (Female)} = (3.5)(\text{BSA})$$

Ventilator frequency is the primary control to regulate the $PaCO_2$.

Adjusting the Frequency. The initial frequency setting of 10 to 12/min and the calculation shown above are based on the assumption that both CO_2 production and physiologic deadspace are normal. If the CO_2 production is elevated (e.g., due to an increase of metabolic rate) or the physiologic deadspace is increased (e.g., due to a decrease of pulmonary perfusion), the minute volume required to normalize the $PaCO_2$ will need to be increased. Since increasing the tidal volume results in higher airway pressures on a volume-limited ventilator, it is usually more appropriate to increase the minute volume by increasing the ventilator frequency.

↑ frequency if the $PaCO_2$ is too high; ↓ frequency if the $PaCO_2$ is too low.

After placing the patient on a ventilator, blood gases should be obtained within 15 to 30 min after the patient has stabilized, to assess both ventilation and oxygenation. Since the $PaCO_2$ varies inversely with the alveolar minute ventilation, a higher than normal $PaCO_2$ (e.g., >45 mm Hg or >50 mm Hg for patients with chronic CO_2 retention) means the patient's minute volume should be increased, usually by increasing the frequency. On the other hand, a lower than normal $PaCO_2$ (e.g., <35 mm Hg or <40 mm Hg for patients with CO_2 retention) indicates that the minute volume should be decreased, usually by decreasing the frequency.

TABLE 8-6 Calculation of Predicted Body Weight

The predicted body weight (PBW) in pounds (lb) and kilograms (kg) can be calculated as follows: Male PBW in lb = 106 + [6 × (Height in inches − 60)] Female PBW in lb = 105 + [5 × (Height in inches − 60)]
Convert the patient's body weight from pounds to kilograms by dividing pounds by 2.2.

Tidal Volume

✓ The initial tidal volume is usually set between 10 and 12 mL/kg of predicted body weight.

The initial tidal volume is usually set between 10 and 12 mL/kg of predicted body weight. Usually the patient's actual weight can be used for selecting the tidal volume unless the patient is significantly underweight or overweight. Table 8-6 shows a method to calculate the patient's predicted body weight.

The lower end of the acceptable tidal volume range (i.e., about 10 mL/kg) may be appropriate for certain patients. Tidal volumes as low as 6 mL per kg of predicted body weight have been recommended for ARDS patients (de Durante et al., 2002). The primary reason for using lower tidal volumes (i.e., permissive hypercapnia) is to minimize the airway pressures and the risk of barotrauma (Feihl et al., 1994). However, use of low tidal volume ventilation may lead to complications such as acute hypercapnia, increased deadspace ventilation and work of breathing, dyspnea, severe acidosis, and atelectasis (Kallet et al., 2001a, 2001b).

✓ Decreasing the tidal volume by 100 to 200 mL in COPD patients reduces the expiratory time requirements and helps to prevent air trapping.

COPD patients may also benefit from a reduced tidal volume setting. These patients have reduced expiratory flow rates due to decreased alveolar elastic recoil. For this reason, a longer expiratory time is needed for complete exhalation. If there is not enough time for complete exhalation, air trapping, V/Q mismatch, hypoxemia, and hypercapnia may result. Decreasing the tidal volume by 100 to 200 mL in COPD patients reduces the expiratory time requirements and helps to prevent air trapping. A higher **flow rate** may also be used to shorten the inspiratory time and lengthen the expiratory time.

flow rate: Peak flow during the inspiratory phase. It determines how fast the tidal volume is delivered to the patient.

For patients with a reduction of lung volumes due to lung resection, lower tidal volumes may also become necessary. Table 8-7 lists examples of clinical conditions where lower tidal volume settings may be beneficial or necessary for the patient.

circuit compressible volume: Expansion of the ventilator circuits during inspiration leading to a small "lost" volume of gas that does not reach the patient, but is recorded as part of the expired tidal volume.

Gas Leakage and Circuit Compressible Volume. The tidal volume actually delivered to the patient's lungs is usually lower than the set tidal volume. This is mainly due to (1) gas leakage in the ventilator circuitry, (2) gas leakage at the endotracheal tube cuff, and (3) **circuit compressible volume** loss.

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TABLE 8-7 Conditions That May Require Lower Tidal Volumes

Condition	Examples
Increase of airway pressure requirement	ARDS Pulmonary edema
Increase of lung compliance	Emphysema
Decrease of lung volumes	Pneumonectomy

When significant gas leakage (>5% of ventilator tidal volume) occurs, the cause must be identified and corrected. Minor gas leakage and circuit compressible volume loss can be compensated by using a larger tidal volume. Some ventilators automatically compensate for the compressible volume loss and thus maintain a stable tidal volume. Other ventilators (e.g., Hamilton Veolar) measure the volume delivered to the patient at the airway opening. This allows detection of significant volume loss due to circuit compression factor or gas leakage.

Ventilator circuits are compliant and expand during a positive pressure breath. The amount of circuit expansion results in a volume that does not reach the patient but is recorded as part of the expired tidal volume. This volume "lost" in the ventilator circuit is called the circuit compressible volume and it may be calculated by following the steps in Table 8-8 (Barnes et al., 1994; Wilkins et al., 2003).

Once the circuit compressible volume is known, the patient's corrected tidal volume can be calculated by:

✓ See Appendix 1 for example.

$$\text{Corrected Tidal Volume} = \text{Expired Tidal Volume} - \text{Circuit Compressible Volume}$$

Pressure Support

Pressure support ventilation (PSV) is used to augment a patient's breathing effort by reducing the airflow resistance during spontaneous breathing. The contributing factors of airflow resistance during mechanical ventilation may include the artificial airway, ventilator circuit, and secretions. Pressure support (PS) is available in modes of ventilation that allows spontaneous breathing (e.g., SIMV). The patient must also be able to breathe spontaneously. The initial pressure support level can be calculated as follows.

$$\text{PS level} = [(\text{PIP} - \text{P}_{\text{plat}})/\dot{V}_{\text{mach}}] \times \dot{V}_{\text{spon}}$$

PS level: Initial pressure support ventilation setting

PIP: Peak inspiratory pressure

P_{plat}: Plateau pressure

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TABLE 8-8 Determination of Circuit Compressible Volume

1. With the circuit warmed to an operating temperature, set the frequency at 10 to 16/min and the tidal volume between 100 and 200 mL with minimal flow rate and maximum high pressure limit.
2. Completely occlude the patient Y-connection of the ventilator circuit.
3. Record the expired volume (mL) and the peak inspiratory pressure during Y occlusion (cm H_2O).
4. Divide the expired volume (mL) by the peak inspiratory pressure during Y occlusion(cm H_2O); this is the circuit compression factor.
5. Multiply the circuit compression factor (mL/cm H_2O) by the peak inspiratory pressure during mechanical ventilation (cm H_2O), or (peak inspiratory pressure–PEEP) if PEEP is used.

Example:

Expired volume = 150 mL; Peak inspiratory pressure (Y occlusion) = 50 cm H_2O;
Peak inspiratory pressure (mechanical ventilation) = 60 cm H_2O; PEEP = 10 cm H_2O.

Circuit compression factor = 150 mL/50 cm H_2O = 3 mL/cm H_2O
Circuit compression volume = 3 mL/cm H_2O × (60 − 10) cm H_2O = 3 × 50 = 150 mL

$\dot{V}_{vent}$: Inspiratory flow of ventilator, in L/min

$\dot{V}_{spon}$: Inspiratory flow during spontaneous breathing in L/min (obtained via flow/time graphic *or* estimated to be 500 mL/sec or 30 L/min)

As shown in the equation, the level of pressure support needed is partly based on the PIP and P_{plat}. For this reason, the PS level must be adjusted on an as-needed basis depending on the changing conditions that alter the PIP and P_{plat}.

For *weaning* from mechanical ventilation with a *spontaneous breathing trial,* PS is titrated until achieving a spontaneous frequency of 20 to 25/min or a spontaneous tidal volume of 8 to 10 mL/kg predicted body weight (PBW). A PSV of greater than 30 cm H_2O is rarely needed since these patients are typically not ready for weaning. For further weaning, the PS level is reduced by 2 to 4 cm H_2O increments as tolerated. Extubation can be considered when the PS level reaches 5 to 8 cm H_2O for 2 hours with no signs of respiratory distress.

F_IO_2

✓ After stabilization of the patient, the F_IO_2 is best kept below 50% to avoid oxygen-induced lung injuries.

For patients with severe hypoxemia or abnormal cardiopulmonary functions (e.g., post-resuscitation, smoke inhalation, ARDS), the *initial* F_IO_2 may be set at 100%. The F_IO_2 should be evaluated by means of arterial blood gas analyses after stabilization of the patient. It should be adjusted accordingly to maintain a PaO_2

between 80 and 100 mm Hg (lower for patients with chronic CO_2 retention). After stabilization of the patient, the F_IO_2 is best kept below 50% to avoid oxygen-induced lung injuries (Shapiro et al., 1994).

For patients with mild hypoxemia or patients with normal cardiopulmonary functions (e.g., drug overdose, uncomplicated postoperative recovery), the initial F_IO_2 may be set at 40% or at the patient's F_IO_2 prior to mechanical ventilation. It must also be evaluated and changed accordingly by means of subsequent blood gas analyses and correlated with pulse oximetry trending.

PEEP

Positive end-expiratory pressure (PEEP) increases the functional residual capacity and is useful to treat refractory hypoxemia (low PaO_2 not responding to high F_IO_2). The initial PEEP level may be set at 5 cm H_2O. Subsequent changes of PEEP should be based on the patient's blood gas results, F_IO_2 requirement, tolerance of PEEP, and cardiovascular responses. For other methods to titrate optimal PEEP, see Table 12-4 ("Titration of optimal PEEP using PaO_2 and compliance as indicators") and Table 15-4 ("Decremental recruitment maneuver (RM) to determine optimal PEEP").

✓ Set the initial PEEP at 5 cm H_2O and make changes based on the patient's blood gas results, F_IO_2 requirement, tolerance of PEEP, and cardiovascular responses.

I:E Ratio

I:E ratio: A time ratio comparing the inspiratory time and expiratory time, normally between 1:2 and 1:4 in mechanical ventilation. This ratio is regulated by the inspiratory flow rate, I time, or E time and is affected by the tidal volume and respiratory rate.

The **I:E ratio** is the ratio of inspiratory time to expiratory time. It is usually kept in the range between 1:2 and 1:4. A larger I:E ratio (longer E ratio) may be used on patients needing additional time for exhalation because of the possibility of air trapping and auto-PEEP. Presence of air trapping during mechanical ventilation may be checked by occluding the expiratory port of the ventilator circuit at the end of exhalation. Auto-PEEP is present when the end-expiratory pressure does not return to baseline pressure (i.e., 0 cm H_2O or the PEEP level when PEEP is in use) at the end of expiration. Presence of auto-PEEP should be apparent on ventilator waveforms (e.g., pressure-time waveform).

✓ Auto-PEEP is present when the end-expiratory pressure does not return to baseline pressure at the end of expiration.

✓ Presence of auto-PEEP should be apparent on ventilator waveforms (e.g., pressure-time waveform).

Inverse I:E ratios have been used to correct refractory hypoxemia in ARDS patients with very low compliance. But it should not be the initial I:E setting since reverse I:E ratio has its inherent cardiovascular complications. Inverse I:E ratio should be tried only after traditional strategies have failed to improve a patient's ventilation and oxygenation status.

Depending on the features available on the ventilator, the I:E ratio may be altered by manipulating any one or a combination of the following controls: (1) flow rate, (2) inspiratory time, (3) inspiratory time %, (4) frequency, and (5) minute volume (tidal volume and frequency).

Effects of Flow Rate on I:E Ratio. Adjusting the flow rate is the most common method to change an I:E ratio because the flow rate control is a feature available on almost all ventilators. Table 8-9 shows the effects of flow rate change on the I time, E time, and I:E ratio when the V_T and f are kept unchanged. Note that the I time and I:E ratio are inversely related. A longer I time leads to a lower I:E ratio (Tejeda et al., 1997).

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TABLE 8-9 Effects of Flow Rate Change on I Time, E Time, and I:E Ratio

Parameter Change	I Time	E Time	I:E Ratio
Increase flow rate	Decrease	Increase	Increase
Decrease flow rate	Increase	Decrease	Decrease

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Other Ventilator Controls That Affect the I:E Ratio. Besides the flow rate control, other settings available on some ventilators may also alter the I:E ratio. Table 8-10 shows the effect tidal volume change on the I:E ratio, and Table 8-11 shows the effect of frequency change on the I time, E time, and I:E ratio.

Changing the I:E Ratio. Since the I:E ratio may be changed by altering different settings available on selected ventilators, different methods to obtain a desired I:E ratio are provided as follows (Chang, 2011).

✓ The calculated flow in Example 1 is the *minimum* flow rate required for this I:E ratio. It is usually set 10 L/min higher to meet changing minute volume requirements.

Example 1 Using Flow to Change the I:E Ratio

Given: Minute Volume = 12 L/min

Desired I:E Ratio = 1:3

Calculate: The flow rate for an I:E ratio of 1:3

Solution: Flow = Minute Volume × Sum of I:E Ratio

= 12 L/min × (1 + 3)

= 12 L/min × 4

= 48 L/min

TABLE 8-10 Effects of V_T Change on I Time, E Time, and I:E Ratio

Parameter Change	I Time	E Time	I:E Ratio
Increase tidal volume	Increase	Decrease	Decrease
Decrease tidal volume	Decrease	Increase	Increase

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TABLE 8-11 Effects of Frequency Change on I Time, E Time, and I:E Ratio

Parameter Change	I Time	E Time	I:E Ratio
Increase f	Minimal change	Decrease	Decrease
Decrease f	Minimal change	Increase	Increase

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At f of 16/min, the I time needed for an I:E ratio of 1:4 is 0.75 sec. The E time is (3.75 − 0.75) sec = 3 sec.

Example 2 Using I Time to Change the I:E Ratio

Given: f = 16/min

Desired I:E Ratio = 1:4

Calculate: The I time needed for an I:E ratio of 1:4

Solution: Since f = 16/min, time for each breath

$$= 60\text{ sec}/16 \text{ or } 3.75\text{ sec}$$

$$\begin{aligned}\text{I Time} &= \text{Time for Each Breath} \times [\text{I Ratio / Sum of I:E Ratio}]\\ &= 3.75\text{ sec} \times [1/(1 + 4)]\\ &= 3.75\text{ sec} \times [1/5]\\ &= 3.75\text{ sec}/5\\ &= 0.75\text{ sec}\end{aligned}$$

I Time % and I:E Ratio. Some ventilators (e.g., Hamilton Veolar) permit the I:E ratio to be preset, usually by setting an I time % (percent inspiratory time). In these ventilators, the flow rate is automatically adjusted by the ventilator to maintain a constant I:E ratio regardless of changes in tidal volume or frequency.

The I time % and I:E ratio equivalent are listed in Table 8-12. For other I:E ratios not listed in the table, they may be calculated by following Example 3:

The I time % needed for an I:E ratio of 1:3.5 is about 22%.

Example 3 Using I Time % to Set the I:E Ratio

Given: Desired I:E Ratio = 1:3.5

Calculate: The I time % needed for an I:E ratio of 1:3.5

Solution:

$$\begin{aligned}\text{I Time \%} &= \frac{\text{I Ratio}}{\text{Sum of I:E Ratio}}\\ &= \frac{1}{(1 + 3.5)}\\ &= \frac{1}{4.5}\\ &= 22\%\end{aligned}$$

Flow Pattern

Most modern ventilators offer different inspiratory flow patterns. Although there are subtle variations, the principal flow patterns are (1) square (constant) flow pattern, (2) accelerating (ascending) flow pattern, (3) decelerating (descending) flow pattern, and (4) sine wave flow pattern. The waveforms for each of these flow patterns are shown in Figure 8-1.

The square flow pattern may be used initially upon setting up the ventilator. This flow pattern provides an even, constant peak flow during the entire inspiratory

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TABLE 8-12 | Time % and I:E Ratio Equivalent

I Time %	I:E Ratio
14.3%	1:6
16.7%	1:5
20%	1:4
25%	1:3
33.3%	1:2
50%	1:1
60%	1.5:1
66.7%	2:1

phase. The initial peak flow at the very beginning of the inspiratory phase should help to overcome the airway resistance and parenchymal elastance, and the remaining peak flow throughout the inspiratory phase should enhance gas distribution in the lungs. Adjustment of the flow pattern may be made after stabilization of the patient. Note that the constant flow pattern is the only flow pattern in which the peak flow rate equals the mean flow rate. All other flow patterns will produce a mean flow rate that is less than the peak flow.

With its increasing flow throughout the respiratory cycle, the accelerating waveform may improve the distribution of ventilation in patients with partial airway obstruction. The decelerating flow pattern typically produces a high initial inspiratory pressure and the decrease in flow may help improve distribution of tidal volume and gas exchange (Campbell et al., 2002; Waugh et al., 2007). For patients with COPD, the decelerating flow may reduce the peak inspiratory pressure, mean airway pressures, physiologic deadspace, and $PaCO_2$ (Yang et al., 2002). The sine

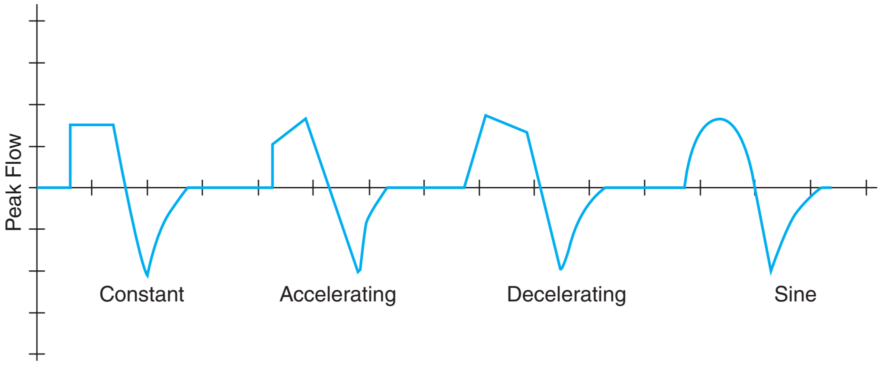

FIGURE 8-1 Normal flow tracing of four different flow patterns: constant, accelerating, decelerating, and sine.

wave flow pattern is considered more physiologic because it is similar to the flow pattern during spontaneous breathing. The sine wave may also improve the distribution of ventilation and therefore improve gas exchange.

For ventilators that do not permit a preset inspiratory time, the inspiratory time may increase if the patient's peak inspiratory pressure increases. This is because as the PIP increases, the pressure gradient between the ventilator and the patient's airway opening increases, resulting in an increased inspiratory time. However, on ventilators in which the inspiratory time is preset, such as the Hamilton Veolar, the inspiratory time is held constant for any flow pattern selected.

In performing calculations that involve the inspiratory flow as a variable (e.g., Resistance = Pressure/Flow), the mean inspiratory flow should be used. Since the only flow pattern in which the peak flow equals the mean inspiratory flow is the square wave pattern, the ventilator should be switched to a constant flow pattern prior to measurement.

VENTILATOR ALARM SETTINGS

Although different ventilators have different alarm systems, the following alarms should be basic to any ventilator: low exhaled volume alarm, low inspiratory pressure alarm, high inspiratory pressure alarm, apnea alarm, high frequency alarm, and F_IO_2 alarm. These alarms should be backed up by a battery source to prevent malfunction in the event of electrical failure.

✓ The *low* exhaled volume alarm (low volume alarm) should be set at about 100 mL *lower* than the expired mechanical tidal volume.

Low Exhaled Volume Alarm

The *low* exhaled volume alarm (low volume alarm) should be set at about 100 mL *lower* than the expired mechanical tidal volume. This alarm is triggered if the patient does not exhale an adequate tidal volume. This alarm is typically used to detect a system leak or circuit disconnection.

✓ The *low* inspiratory pressure alarm (low pressure alarm) should be set at 10 to 15 cm H_2O *below* the observed peak inspiratory pressure.

Low Inspiratory Pressure Alarm

The *low* inspiratory pressure alarm (low pressure alarm) should be set at 10 to 15 cm H_2O *below* the observed peak inspiratory pressure. This alarm is triggered if the peak inspiratory pressure is less than the alarm setting. The low inspiratory pressure alarm complements the low exhaled volume alarm and is also used to detect system leaks or circuit disconnection.

✓ The *high* inspiratory pressure alarm (high pressure limit alarm) should be set at 10 to 15 cm H_2O *above* the observed peak inspiratory pressure.

High Inspiratory Pressure Alarm

The *high* inspiratory pressure alarm (high pressure limit alarm) should be set at 10 to 15 cm H_2O *above* the observed peak inspiratory pressure. This alarm is triggered when the peak inspiratory pressure is equal to or higher than the high pressure limit. Once the alarm is triggered by airflow obstruction, inspiration is immediately terminated and the ventilator goes into expiratory cycle.

The patient must be evaluated to determine the cause of the airflow obstruction. Common causes that trigger the high inspiratory pressure alarm include water in the ventilator circuit, kinking or biting of the endotracheal tube, secretions in the airway, bronchospasm, mucus plugs, tension pneumothorax, decreases in lung compliance, increases in airway resistance, and coughing.

Apnea Alarm

The apnea low volume and low pressure alarms are triggered in apnea and circuit disconnection (i.e., inadvertent disconnection or during endotracheal suctioning). Inadvertent circuit disconnection is a common event in patients with a tracheostomy tube due to lack of allowance for airway flexibility as with an endotracheal tube. The apnea alarm should be set with a 15- to 20-sec time delay, with less time delay at higher frequency. On some ventilators, the apnea alarm also triggers an apnea backup ventilation mode in which the ventilator provides ventilatory support until the alarm condition no longer exists.

✓ The apnea alarm should be set with a 15- to 20-sec time delay.

High Frequency Alarm

✓ The *high* frequency alarm should be set at 10 /min *over* the observed frequency.

The *high* frequency alarm should be set at 10/min *over* the observed frequency. Triggering of the high frequency alarm may indicate that the patient is experiencing respiratory distress. See Chapter 12, “Management of Mechanical Ventilation,” for a discussion on the causes and management of the high frequency alarm.

High and Low F_IO_2 Alarms

✓ Set the low and high F_IO_2 alarms at 40% and 50%, respectively, for an F_IO_2 of 45%.

The high F_IO_2 alarm should be set at 5% to 10% over the analyzed F_IO_2 and the low F_IO_2 alarm should be set at 5% to 10% below the analyzed F_IO_2.

HAZARDS AND COMPLICATIONS

Mechanical ventilation has many potential hazards (e.g., ventilator disconnection, nosocomial infection) and complications (e.g., barotrauma, hypotension). The frequency of occurrence is directly related to the length of mechanical ventilation (Liu et al., 1991; Pierson, 1990). Patients who require mechanical ventilation for longer periods are likely to develop more complications.

This section summarizes the common hazards and complications of mechanical ventilation based on several prospective studies. Special emphasis is provided for barotrauma and decrease in cardiac output and blood pressure since the effects of these complications are more urgent to the patient’s progress and outcome.

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Types of Hazards and Complications

Prospective studies of patient outcome during mechanical ventilation indicate that hazards and complications are related to (1) positive pressure ventilation (e.g., barotrauma, impedance of cardiac output), (2) patient condition (e.g., organ failure), (3) ventilator and artificial airway (accidental extubation), and (4) medical professionals (e.g., nosocomial infection). Examples for each of these four areas are shown in Table 8-13.

TABLE 8-13 Hazards and Complications of Mechanical Ventilation

Condition	Examples
Related to positive pressure ventilation	Barotrauma (pneumothorax, mediastinal air leak, subcutaneous air leak) Hypotension, decrease in cardiac output Arrhythmia Oxygen toxicity Bronchopleural fistula Bronchopulmonary dysplasia (in infants) Upper gastrointestinal hemorrhage
Related to patient condition	Infection (due to reduced immunity) Physical and psychologic trauma Multiple organ failure (may be preexisting)
Related to equipment (ventilator and artificial airway)	Ventilator and alarm malfunction Ventilator circuit disconnection Accidental extubation Main bronchus intubation Postintubation stridor Endotracheal tube blockage Tissue damage Atelectasis (due to inadequate tidal volume)
Related to medical professionals	Nosocomial pneumonia (due to cross-contamination) Inappropriate ventilator settings Misadventures (due to lapses of understanding and communication)

(Data from Ventilator alarm failures, Bourke et al., 1987; Milligan, 1992; Pryn et al., 1989; Slee et al., 1988; and Other hazards and complications: Bezzant et al., 1994; Cox et al., 1991; Liu et al., 1991; Pierson, 1990; Rivera et al., 1992.)

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Malfunction and Misuse of Alarms

The Joint Commission (TJC) reviewed 23 reported deaths or injuries related to long-term mechanical ventilation and found 19 deaths and four in coma. Sixty-five percent of the deaths or injuries were related to the malfunction or misuse of ventilator alarms. A breakdown of the causes revealed that the alarms were either turned off or set incorrectly, no alarm was available for certain disconnections, testing of alarms was not performed, or response to alarm was delayed or absent (NYSNA, 2002).

These tragedies could be prevented by implementing regular preventive maintenance and testing of alarm systems on ventilators and monitors. The alarms must also be sufficiently audible with respect to the room design, distance, and noise level of the immediate patient care area. Over dependence on alarms should be avoided and emphasis should be placed on frequent direct observation and assessment of the patient-ventilator system.

Barotrauma

✔ Risk of barotrauma is high when PIP >50 cm H_2O, plateau pressure >35 cm H_2O, mPaw >30 cm H_2O, and PEEP >10 cm H_2O.

Barotrauma is the term used to describe lung tissue injury or rupture that results from the shearing force of alveolar over distention. General agreement is that in most cases, peak inspiratory pressures greater than 50 cm H_2O, plateau pressures greater than 35 cm H_2O, mean airway pressures greater than 30 cm H_2O, and PEEP greater than 10 cm H_2O may induce the development of barotrauma (Bezzant et al., 1994; Slutsky, 1994). The risk of barotrauma also increases with the duration of positive pressure ventilation.

Barotrauma can occur at mean airway pressures lower than 30 cm H_2O either due to patient susceptibility or due to an uneven distribution of ventilation. COPD patients are more susceptible to barotrauma presumably due to air trapping and weakened parenchymal areas (e.g., lung blebs and bullae). Uneven distribution of ventilation may result in patients with significant airway obstruction and lung parenchymal changes. A mechanical tidal volume tends to preferentially distribute to areas of low resistance and high compliance during the early portion of inspiration. This may result in transient elevated alveolar pressures with resultant over distention and rupture despite what would normally be accepted as a “safe” pressure.

Other lung injuries that may occur as a result of positive pressure ventilation include pulmonary interstitial emphysema, pneumomediastinum, pneumoperitoneum, pneumothorax, tension pneumothorax, and subcutaneous emphysema.

Decrease in Cardiac Output and Blood Pressure

Positive pressure ventilation has been implicated in the development of decreased cardiac output and arterial blood pressure (Bezzant et al., 1994; Franklin et al., 1994). The reason is that positive airway and alveolar pressures may potentially increase the normally subatmospheric pleural pressures that surround the heart and vena cava. The increased pleural pressure tends to compress the right atrium and vena

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cava, increasing the intravascular resistance and pressures associated with these structures. The pressures in the vena cava and right atrium are approximately equal and are collectively known as the central venous pressure (CVP). The usually low central venous pressure creates an intravascular pressure gradient between the right atrium and the systemic venous drainage that augments venous blood return to the right atrium. If positive pressure ventilation increases the CVP, the pressure gradient between the right atrium and the venous drainage will be decreased with a resultant decreased venous return to the right atrium. If the venous return is significantly reduced, this can result in a decreased cardiac output and arterial hypotension.

Since positive pressure ventilation increases the CVP, the pressure gradient between the right atrium and the venous drainage will be decreased with a resultant decreased venous return to the right atrium.

If the patient has no preexisting cardiovascular disease and is not hypovolemic, a competent cardiovascular system can compensate for a small drop in venous return and thus maintain cardiac output and blood pressure. The two primary compensatory mechanisms include an increased heart rate and arterial vasoconstriction initiated by the cardiac baroreceptors.

A competent cardiovascular system can compensate for a small drop in venous return by an increased heart rate and arterial vasoconstriction.

The magnitude of the increase in the CVP and the resultant decrease in venous return depends on several factors, including the airway pressure, lung compliance, and chest wall compliance. Higher airway pressures will more likely result in higher pleural and central venous pressures. It is important to note that increases of the mean airway pressures tend to depress venous return more than increases of the peak inspiratory pressures.

The degree of increased pleural pressure for a given airway pressure is further affected by the patient's lung and chest wall compliance. If the patient's lung compliance is low (stiff lungs), then airway pressures are less readily transmitted into the pleural space. Therefore in patients with low lung compliance, a given airway pressure will result in a smaller increase in pleural pressure and a less dramatic decrease in venous return. This does not mean that patients with low lung compliance cannot have significant decreases in cardiac output due to positive pressure ventilation. These patients must also be closely monitored for potentially significant decreases in cardiac output and blood pressure.

Patients with more compliant lungs, such as COPD patients, tend to more readily transmit a higher airway pressure into the pleural space. Therefore in these patients, a given airway pressure will tend to result in a more dramatic decrease in venous return and cardiac output.

High airway pressures are more detrimental to the cardiac output in patients with high lung compliance than those with low compliance.

The effects of chest wall compliance on the transmission of airway pressure into the pleural space are exactly opposite to the effects of lung compliance. A low chest wall compliance (stiff chest wall) will tend to increase the pleural pressure more significantly for a given airway pressure than a normal chest wall compliance. Conditions in which the chest wall would be less compliant than normal include the application of tight chest wall bandages that encircle the thorax and extensive chest wall burn injuries.

All ventilator patients must be monitored for signs of cardiovascular instability. However, because ventilator patients with suspected or known preexisting cardiovascular disease are more likely to suffer clinically significant decreases in cardiac output and blood pressure, these patients must be monitored with an extra measure of vigilance.

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SUMMARY

Initiation of mechanical ventilation requires many decisions, ranging from the mode of ventilation to the level of PEEP. Nevertheless, this process can be simplified by following a set of established guidelines. Under most clinical conditions, the initial settings on the ventilator will satisfy the immediate requirement for most patients.

It is essential to remember that the initial settings are just that; subsequent changes to these settings must be made based on the ever-changing condition of the patient. In summary, Table 8-14 outlines the indications for mechanical ventilation and Table 8-15 provides an overview of the initial ventilator settings for an adult patient.

TABLE 8-14 Indications for Mechanical Ventilation

Indication	Parameters
Acute ventilatory failure	$PaCO_2 > 50$ mm Hg (Higher for COPD) pH < 7.30 Apnea
Impending ventilatory failure	Tidal volume < 3 to 5 mL/kg Frequency > 25 to 35/min Minute ventilation > 10 L/min Vital capacity < 15 mL/kg MIP < -20 cm H_2O Rising $PaCO_2 > 50$ mm Hg
Severe hypoxemia	$PaO_2 < 60$ mm Hg at $F_1O_2 > 50\%$, or $PaO_2 < 40$ mm Hg at any F_1O_2 PaO_2/FIO_2 (P/F ratio): ≤ 300 mm Hg for ALI, ≤ 200 mm Hg for ARDS
Prophylactic ventilatory support	Reduce risk of pulmonary complications Prolonged shock Head injury Smoke inhalation Reduce hypoxia of major body organs Hypoxic brain Hypoxia of heart muscles Reduce cardiopulmonary stress Prolonged shock Coronary artery bypass surgery Other thoracic or abdominal surgeries

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TABLE 8-15 Initial Ventilator Settings

Parameter	Setting	Notes
Mode	Assist/Control or SIMV	Provide ventilatory support.
f	10 to 12/min	Primary control to regulate ventilation. Guided by $PaCO_2$.
V_T	10 to 12 mL/kg	Peak inspiratory pressure is *directly* related to the V_T setting.
		Use lower V_T (e.g., 6 to 8 mL/kg) to reduce risk of pressure-related lung injuries.
F_IO_2	100% for severe hypoxemia or compromised cardiopulmonary status	40% for mild hypoxemia or normal cardiopulmonary status.
PEEP	$\geq$5 cm H_2O for refractory hypoxemia	Monitor patient and note cardiovascular adverse effects.
I:E	1:2 to 1:4	1:4 for patients needing longer E time due to air trapping.
Flow pattern	(Constant)	Other flow patterns for a lower peak inspiratory pressure (manometer) and better gas distribution (breath sounds).

Self-Assessment Questions

1. Which of the following is *not* an indication for mechanical ventilation?

   A. acute ventilatory failure
   B. impending ventilatory failure
   C. severe hypoxemia
   D. airway obstruction

2. A patient has an admitting diagnosis of acute ventilatory failure. This condition is characterized by a $PaCO_2$ of ________ mm Hg or greater with an accompanying respiratory ________.

   A. 20, acidosis
   B. 20, alkalosis
   C. 50, acidosis
   D. 50, alkalosis

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3. Impending ventilatory failure may be evaluated by trending a patient's:

   A. respiratory parameters and mechanics.
   B. arterial PO_2.
   C. vital signs.
   D. hemodynamic parameters.

4. Among other criteria of assessment, impending ventilatory failure may be present when the patient's minute ventilation is _______ 10 L/min and maximum inspiratory pressure is less than _______.

   A. more than, 20 cm H_2O
   B. more than, -20 cm H_2O
   C. less than, 20 cm H_2O
   D. less than, -20 cm H_2O

5. The primary purposes of prophylactic mechanical ventilation include all of the following *except*:

   A. to minimize the risk of pulmonary complications.
   B. to reduce prolonged hypoxia of major body organs.
   C. to reduce the work of the cardiopulmonary system.
   D. to monitor the arterial blood gases and vital signs.

6. A physician asks the therapist to set up a ventilator using volume-controlled made for a 35-year-old postoperative patient who weighs 132 lb (60 kg). The therapist should use an initial tidal volume of _______ and frequency of _______.

   A. 400 mL, 12/min
   B. 400 mL, 20/min
   C. 600 mL, 20/min
   D. 600 mL, 12/min

7. A therapist is using the standard procedure to measure the compressible volume of a new ventilator circuit. The observed expired volume (mL) and peak inspiratory pressure (cm H_2O) are 150 mL and 50 cm H_2O, respectively. The circuit compressible evaluated is therefore:

   A. 2 mL/cm H_2O.
   B. 4 mL/cm H_2O.
   C. 5 mL/cm H_2O.
   D. 3 mL/cm H_2O.

8. With a circuit compressible volume of 3 mL/cm H_2O, what would be the delivered tidal volume with an expired tidal volume of 550 mL and a peak inspiratory pressure of 30 cm H_2O?

   A. 500 mL
   B. 460 mL
   C. 620 mL
   D. 540 mL

9. Positive end-expiratory pressure (PEEP) may be indicated in patients with decreased ________ and presence of ________.

    A. tidal volume, chronic hypercapnia
    B. functional residual capacity, refractory hypoxemia
    C. vital capacity, acute hypercapnia
    D. tidal volume, refractory hypoxemia

10. At constant tidal volume and frequency, increasing the inspiratory flow rate will lead to a ________ inspiratory time (I time) and ________ expiratory time (E time).

    A. longer, longer
    B. longer, shorter
    C. shorter, longer
    D. shorter, shorter

11. What should be the *minimum* flow rate for a minute volume of 12 L/min and an I:E ratio of 1:4?

    A. 40 L/min
    B. 50 L/min
    C. 60 L/min
    D. 70 L/min

12. If the desired I:E ratio is 1:3, what should be the I time %?

    A. 25%
    B. 40%
    C. 35%
    D. 20%

13. The ________ and ________ are two alarms that are to detect circuit disconnection.

    A. low pressure, high pressure
    B. high PEEP, high volume
    C. low volume, low PEEP
    D. low pressure, low volume

14. Which of the following is *not* a common potential complication of positive pressure ventilation?

    A. hypertension
    B. decrease in cardiac output
    C. accidental patient disconnection
    D. barotrauma

15 to 18. Match the conditions of mechanical ventilation with respective examples of the hazards and complications of mechanical ventilation.

Condition	**Examples**
15. Related to pressure and volume	A. Circuit disconnection
16. Related to patient condition	B. Barotrauma
17. Related to equipment	C. Inappropriate ventilator settings
18. Related to medical professionals	D. Physical and psychologic trauma

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Answers to Self-Assessment Questions

1. D.
2. C.
3. A.
4. B.
5. D.
6. D.
7. D.
8. B.
9. B.
10. C.
11. C.
12. A.
13. D.
14. A.
15. B.
16 D.
17. A.
18. C.

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Chapter 9

Monitoring in Mechanical Ventilation

Walter C. Chop
David W. Chang

Outline

- Introduction
- Vital Signs
  - *Heart Rate*
  - *Blood Pressure*
  - *Respiratory Frequency*
  - *Temperature*
- Chest Inspection
  - *Chest Movement*
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  - *Imaging*
- Fluid Balance and Anion Gap
  - *Fluid Balance*
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  - *Assessment of Oxygenation Status*
  - *Limitations of Blood Gases*
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  - *Capnography Waveforms and Clinical Application*
  - *P(a-et)CO_2 Gradient*
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- Transcutaneous Blood Gas Monitoring
  - *Transcutaneous PO_2 ($PtcO_2$)*
  - *Transcutaneous PCO_2 ($PtcCO_2$)*
- Cerebral Perfusion Pressure
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References

Key Terms

anion gap
cerebral perfusion pressure (CPP)
diffusion defect
dyshemoglobins
end-tidal carbon dioxide monitoring
hypoventilation
hypoxemia
hypoxia
intrapulmonary shunting
oliguria
pulse oximeter
transcutaneous PCO_2 ($PtcCO_2$)
transcutaneous PO_2 ($PtcO_2$)
ventilation/perfusion (V/Q) mismatch

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Name 4 vital signs and list their clinical application.
- Describe the findings of normal and abnormal chest movement, breath sounds, and chest imaging.
- Evaluate fluid balance and anion gap.
- Describe the application and limitations of blood gases, oximetry, and CO-oximetry.
- Describe the application and limitations of capnography.
- Describe the application of transcutaneous blood gas monitoring.
- Calculate cerebral perfusion pressure and explain why a minimal pressure is required for critically ill patients.

INTRODUCTION

Monitoring a patient's clinical condition during mechanical ventilation is vital because clinical status often changes rapidly and unpredictably. A patient's clinical status may be affected by the underlying illness, medications, organ failure, and even the settings on the ventilator.

There are four reasons for monitoring a patient on a continuous basis: (1) baseline measurements can be used to establish the initial treatment plan and serve as a reference point for future measurements; (2) a trend can be established to document the progress or regression of a patient's condition; (3) treatment plans can be added, altered, or discontinued according to the measurements obtained; and (4) high-limit and low-limit alarms can be set on most monitors to safeguard a patient's safety.

This chapter provides an overview of monitoring techniques pertinent to mechanical ventilation.

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VITAL SIGNS

Vital signs (heart rate, blood pressure, respiratory rate, and temperature) can provide very useful information on the overall condition of a patient. During mechanical ventilation, changes in vital signs often indicate changes in the patient's cardiopulmonary status.

Heart Rate

In the intensive care unit, heart rate assessment is readily available on the electrocardiograph (ECG) monitor. High and low alarms can be set on the monitor to warn of tachycardia and bradycardia.

Normal adult heart rate is between 60 and 100/min.

Tachycardia. Tachycardia in adults is defined as a heart rate higher than 100/min. During mechanical ventilation, some conditions that may increase a patient's heart rate are **hypoxemia**, pain, anxiety and stress, fever, drug reactions, and myocardial infarction. Tachycardia can alert the clinician to blood volume or cardiac output deficits. Increase in heart rate must be evaluated as part of the larger picture as it can be secondary to extreme conditions ranging from hypovolemia to anxiety.

Tachycardia may be caused by hypoxemia, hypovolemia, pain, anxiety and stress, fever, drug reactions, and myocardial infarction.

hypoxemia: Deficiency of oxygen in blood; low PaO_2.

Bradycardia. Bradycardia in adults is defined as a heart rate lower than 60/min. Bradycardia often occurs with vagal stimulation during endotracheal suctioning. Preoxygenation is often necessary to minimize the occurrence of arterial desaturation and arrhythmias (e.g., premature ventricular contraction) during suctioning. Since arterial desaturation occurs in as little as 5 sec during suctioning, **hypoxia** and cardiac complications can happen rather rapidly. When arrhythmia or bradycardia occurs, endotracheal suctioning must be stopped and 100% oxygen should be provided to the patient immediately (Burton et al., 1997).

hypoxia: Deficiency of oxygen in tissues.

Bradycardia, if it appears together with a low cardiac output, can be ominous and may suggest a decrease in coronary blood flow. Table 9-1 outlines the conditions that affect the heart rate.

Blood Pressure

Continuous blood pressure monitoring in the critically ill patient is usually done via an indwelling arterial catheter interfaced with a pressure monitor. The most common insertion site for the catheter is the radial artery. Other sites include the brachial, femoral, popliteal, and dorsalis pedis arteries.

Hypertension. Hypertension, higher than normal blood pressure, may be caused by acute or chronic patient conditions. Acute conditions such as fluid overload, vasoconstriction, stress, anxiety, and pain may lead to hypertension. Patients who have a history of congestive heart failure (CHF), cardiovascular disease, or polycythemia may develop hypertension, which can become a complicating factor during mechanical ventilation.

Fluid overload, vasoconstriction, stress, anxiety, and pain may lead to hypertension.

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TABLE 9-1 Conditions That Affect the Heart Rate

Conditions That May Cause Tachycardia	Conditions That May Cause Bradycardia
Hypoxemia	Sudden hypoxia and/or vagal stimulation during endotracheal suctioning
Hypovolemia	Inadequate coronary blood flow
Pain	Heart block
Anxiety and stress	Abnormal SA node function
Fever	Hypothermia
Drug reaction (e.g., epinephrine)	Drug reaction (e.g., morphine sulfate)
Myocardial infarction	

✓ Hypovolemia, positive pressure ventilation, and pump failure are conditions that may cause hypotension.

Hypotension. Hypotension, lower than normal blood pressure, may be due to absolute hypovolemia (blood loss), relative hypovolemia (shock), or pump failure (CHF). When hypotension occurs during mechanical ventilation, it is often associated with excessive intrathoracic pressure, peak inspiratory pressure, and lung volume. Hypotension is one of the complications of positive pressure ventilation or positive end-expiratory pressure. Table 9-2 outlines the conditions that affect the blood pressure.

Respiratory Frequency

The normal spontaneous respiratory frequency for adults is 10 to 16 breaths per minute. An increased respiratory frequency (tachypnea) may be an early warning sign of

TABLE 9-2 Conditions That Affect the Blood Pressure

Conditions That May Cause Hypertension	Conditions That May Cause Hypotension
Fluid overload	Decreased venous return due to positive pressure ventilation
Stress	Absolute hypovolemia (e.g., blood loss, dehydration)
Anxiety	Relative hypovolemia (e.g., sepsis, shock)
Pain	Pump failure (e.g., CHF)
Congestive heart failure	
Cardiovascular disease	
Polycythemia (↑ blood viscosity)	

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hypoventilation: Below normal level of alveolar ventilation characterized by an elevated $PaCO_2$.

hypoventilation or hypoxia. In normal individuals, maximum response to hypoxia occurs below a PaO_2 of 50 mm Hg (West, 2011). If the frequency exceeds 20 breaths per minute and is rising, the patient should be evaluated for the cause of tachypnea.

Tachypnea may precede the development of respiratory failure and use of mechanical ventilation (Krieger & Ershowshy, 1994). During mechanical ventilation, tachypnea is indicative of respiratory dysfunction (Gravelyn & Weg, 1980). When tachypnea and low tidal volume are observed in a patient, successful weaning from mechanical ventilation is not likely (Tobin et al., 1986).

Routine monitoring of a patient's spontaneous respiratory frequency is a simple and very useful method to assess the pulmonary status of a ventilator patient. This is especially true during the weaning process. A sudden increase in spontaneous respiratory frequency during the weaning attempt is indicative of moderate or severe respiratory insufficiency or hypoxia.

✓ Rapid, shallow breathing is a reliable sign of ventilatory insufficiency.

A patient's respiratory frequency can be monitored noninvasively and continuously by an easy-to-use monitoring system. Masimo Rainbow Acoustic Monitoring™ measures the respiratory frequency using an adhesive sensor with an integrated acoustic transducer applied to the patient's neck. A respiratory frequency monitor may be indicated for patients in the postanesthesia setting and for those receiving partial ventilatory support.

Temperature

In the intensive care unit, a patient's temperature may be measured routinely at regular intervals or monitored continuously via a rectal, esophageal, or pulmonary artery catheter probe.

✓ Hyperthermia causes a lower oxygen saturation at any PaO_2.

Hyperthermia. Hyperthermia can occur as a result of infection, tissue necrosis, leukemia, or other conditions that increase a patient's metabolic rate and oxygen utilization. Hyperthermia also shifts the oxyhemoglobin dissociation curve to the right, causing a lower oxygen saturation level at any PaO_2. This oxygen desaturation occurs because increased temperature promotes unloading of oxygen from hemoglobin to the tissues.

✓ Hypothermia lowers a person's basal metabolic rate.

Hypothermia. Hypothermia, though seen less commonly in the critically ill patient, can occur as a result of central nervous system (CNS) problems, metabolic disorders, and from certain drugs or toxins. Hypothermia is sometimes induced in head trauma patients as a means of decreasing the patient's basal metabolic rate.

Hypothermia is also induced in patients undergoing coronary artery bypass (CAB) surgery. At this extreme low temperature, the hypothermic condition must be taken into account during management of the ventilator and patient. For example, the measured PaO_2 and $PaCO_2$ values are higher than the actual values when the sample is collected under hypothermic conditions, but is analyzed at body temperature. In order to have blood gas values that accurately reflect a patient's true ventilatory and oxygenation status, corrections to the patient's core temperature should be done during blood gas analysis.

In other nonextreme hypothermic conditions ($\pm 2°C$), temperature corrections are not necessary as long as the uncorrected PaO_2 is above 60 mm Hg (Malley, 1990).

TABLE 9-3 Conditions That Affect the Body Temperature

Conditions That May Cause Hypothermia	Conditions That May Cause Hyperthermia
Infection	CNS problem
Tissue necrosis	Metabolic disorders
Leukemia	Drugs and toxins
Increased metabolic rate	Induced coronary bypass surgery
	Head injury

✓ Excessive cooling of the phrenic nerve during CAB surgery may cause paralysis of the hemidiaphragms.

Excessive cooling of the phrenic nerve during CAB surgery may cause paralysis of the hemidiaphragms. This condition, though temporary, may take months to resolve completely (Wilkins & Dexter, 1998). Table 9-3 outlines the conditions that affect the body temperature.

CHEST INSPECTION

Since the lungs are protected by the thoracic cage, they are not readily accessible for direct examination. Chest inspection uses indirect methods to assess and evaluate the lungs and related structures.

Chest Movement

During mechanical ventilation it is important to observe the overall chest movement. One should observe the symmetrical movement of the chest with each inspiration as well as the depth and rhythm of each tidal volume cycle.

Asymmetrical movement can occur in conditions such as right main-stem (bronchial) intubation, atelectasis, and tension pneumothorax (Figure 9-1). If dysynchronous

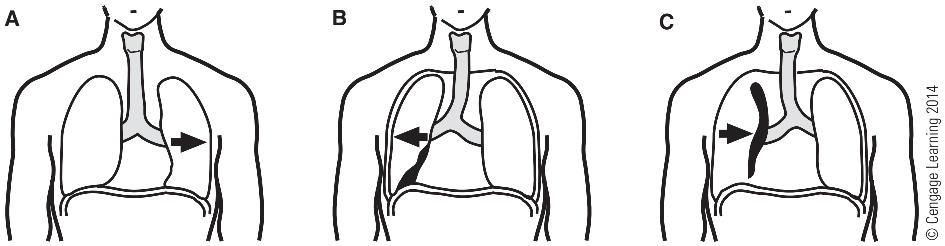

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FIGURE 9-1 Asymmetrical chest movement due to (A) right bronchial intubation; note overinflation of the right lung and underinflation of the left lung; (B) atelectasis, the trachea and mediastinum are shifted to the affected side; (C) tension pneumothorax, the trachea and mediastinum are shifted to the opposite unaffected side.

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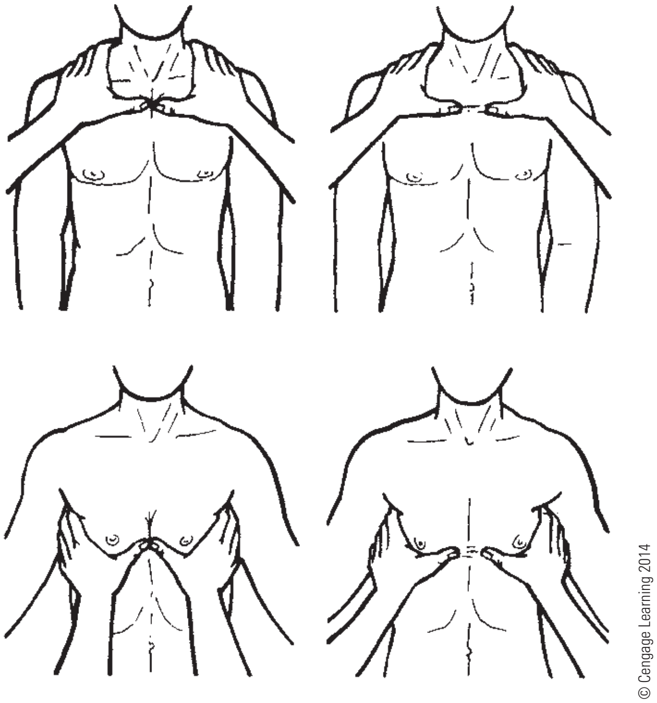
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FIGURE 9-2 Hand positions for assessing symmetry of the chest.

motion of the chest and abdomen is noted, the patient should be evaluated for diaphragmatic fatigue or underlying pathology.

✔ Chest expansion is *symmetrical* when the patient takes in a deep breath and the hands of the examiner move apart in *equal distance from midline*.

Assessment of symmetry of chest excursion or expansion can be done by placing the hands on corresponding opposite position of the right and left sides of the chest or posterior thorax, with the examiner's thumbs touching (Figures 9-2 and 9-3). When the patient takes in a deep breath, the hands of the examiner should move apart in equal distance from midline. If one hand moves less than the other, the

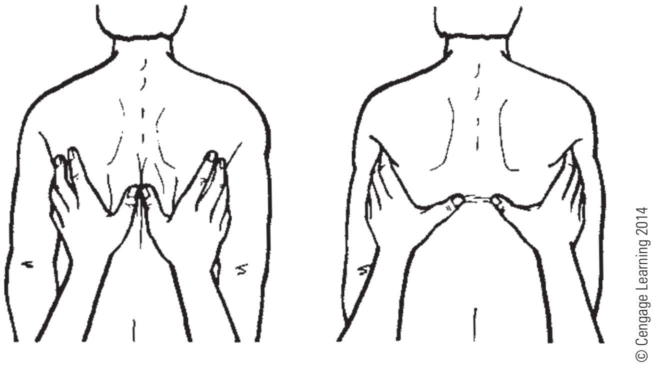
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FIGURE 9-3 Hand positions for assessing symmetry of the posterior thorax.

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TABLE 9-4 Abnormal Breath Sounds and Related Conditions

Breath Sound	Conditions
Diminished or absent	Airway obstruction Atelectasis Main-stem intubation Pleural effusion Pneumothorax
Wheezes	Airway narrowing
Inspiratory crackles	Lung consolidation Pulmonary edema
Coarse crackles	Excessive secretions

affected side with less air movement may have conditions such as consolidation, pleural effusion, atelectasis, and pneumothorax (White, 2003).

Auscultation

Auscultation of a patient's breath sounds should be performed each time the practitioner assesses the patient/ventilator system. Diminished or absent breath sounds or the presence of wheezes and crackles are signs of ventilatory problems and should be recognized as causes of respiratory distress (Wilkins et al. 1998). Table 9-4 shows these abnormal breath sounds and their related clinical conditions.

✓ A side-to-side technique of chest auscultation allows comparison of the quantity of breath sounds between the left and right lungs.

Chest auscultation should be done in a systemic fashion. The quality and quantity of breath sounds should be assessed by placing the stethoscope diaphragm from the left to the right side of the chest (Figures 9-4 and 9-5) (White, 2003). This technique of chest auscultation allows comparison of the quantity of breath sounds. Prior to the procedure, the patient should be instructed to take in a slow, deep breath each time the stethoscope diaphragm touches and rests on the skin. This allows the therapist to concentrate on listening without repeating the same instruction throughout the procedure.

Figures 9-6 through 9-8 show the surface projections of lung segments, and they are helpful for the correct placement of the stethoscope diaphragm. Proper identification of the lung segments involved in the disease process is essential for consistent charting and reporting, and for performing the correct chest percussion and postural drainage procedures.

✓ A cuff leak may be present if distinct air movement can be heard toward the end of a mechanical breath.

The stethoscope can also be used for detection of a leaky cuff on an endotracheal or tracheostomy tube, as well as for right main-stem intubation. A cuff leak may be detected by placing the stethoscope diaphragm over the trachea and on top of the

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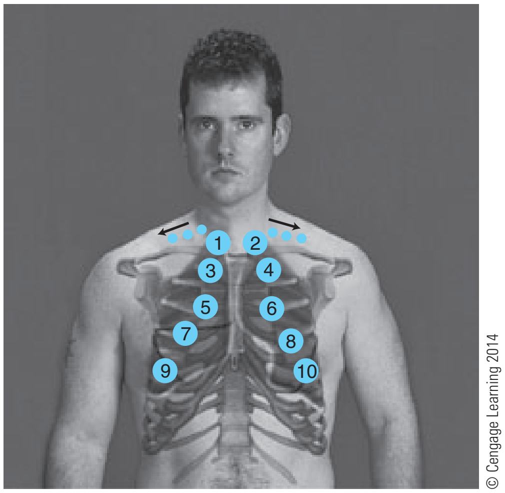
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FIGURE 9-4 Anterior positions for chest auscultation.

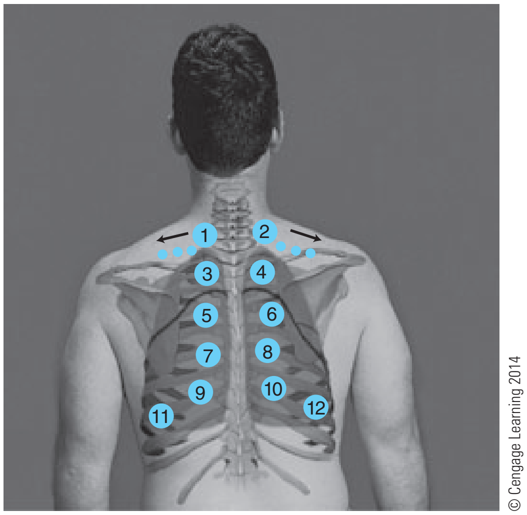
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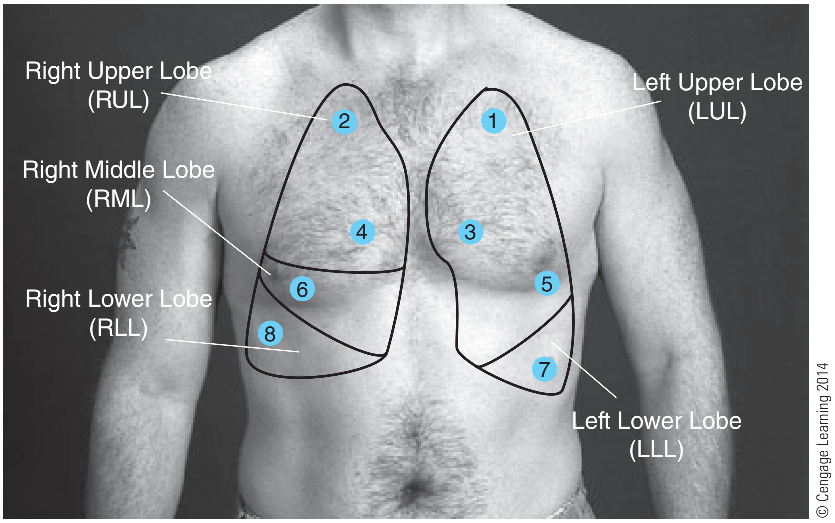
FIGURE 9-5 Posterior positions for chest auscultation.

cuff's location. The cuff is leaking if distinct air movement can be heard toward the end of a mechanical breath. Uneven bilateral breath sounds in the absence of lung pathology (e.g., atelectasis, tension pneumothorax) may indicate main-stem intubation. The placement of an endotracheal tube should be confirmed by chest radiograph.


Right Upper Lobe (RUL)
Left Upper Lobe (LUL)
Right Middle Lobe (RML)
Right Lower Lobe (RLL)
Left Lower Lobe (LLL)
1
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4
5
6
7
8
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FIGURE 9-6 Anterior surface projection of the lung lobes and segments. Left lung: (1) LUL apical segment, (3) LUL anterior segment, (5) LUL lingula inferior and superior segments, (7) LLL anterior segment. Right lung: (2) RUL apical segment, (4) RUL anterior, (6) RML medial and lateral segments, (8) RLL anterior segment.

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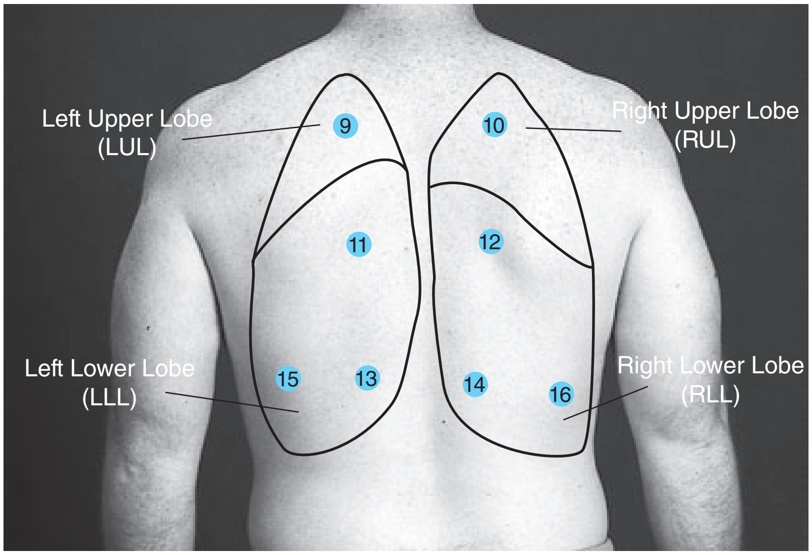
Left Upper Lobe
(LUL)
Right Upper Lobe
(RUL)
Left Lower Lobe
(LLL)
Right Lower Lobe
(RLL)
9
10
11
12
15
13
14
16

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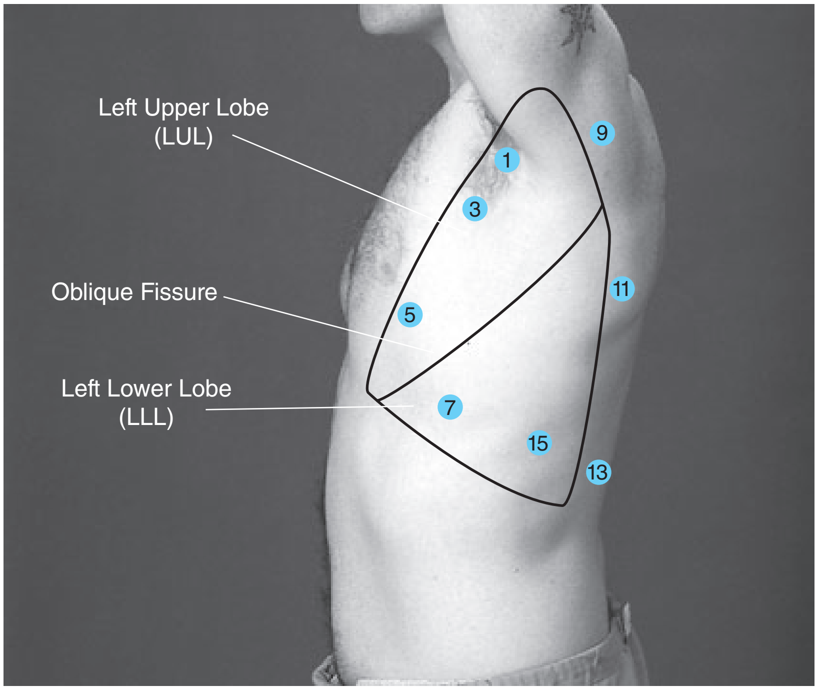
FIGURE 9-7 Posterior surface projection of the lung lobes and segments. Left lung: (9) LUL posterior segment, (11) LLL superior segment, (13) LLL posterior segment, (15) LLL lateral segment. Right lung: (10) RUL posterior segment, (12) RLL superior segment, (14) RLL posterior segment, (16) RLL lateral segment.


Left Upper Lobe
(LUL)
Oblique Fissure
Left Lower Lobe
(LLL)
9
1
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11
5
7
15
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FIGURE 9-8 Left lateral surface projection of the lung lobes and segments. Left upper lobe: (1) LUL apical segment, (3) LUL anterior segment, (5) LUL lingula inferior and superior segments, (9) LUL posterior segment. Left lower lobe: (7) LLL anterior segment, (11) LLL superior segment, (13) LLL posterior segment, (15) LLL lateral segment.

Imaging

The chest radiograph is the most common method to evaluate the conditions of the thoracic structure, lungs, pleural space, inserted catheters, lines, and tubes. Interpretation of the chest radiograph is beyond the scope of this section. Readers are encouraged to use additional resources on the fundamentals and clinical application of chest radiography. This section reviews the normal posterior-anterior (PA) and lateral chest radiographs, the major anatomical structures on the chest radiograph, and the primary reason for using a lateral chest radiograph.

Figure 9-9 shows the PA radiographic image of a normal chest and the corresponding anatomical structures. Figure 9-10 shows the lateral view and the corresponding anatomical structures (White, 2003). Note that the air-filled structures (e.g., trachea, lung parenchyma) are dark-shaded (over exposure of the "negative" film, because air does not absorb X-ray). On the other hand, the tissues and bones (e.g., aortic arch, clavicles) appear to be almost white in shade (under exposure of the "negative" film due to high absorption of the X-ray by the tissues and bones).

In clinical practice, deviations from the normal characteristics of the chest radiograph require further evaluation. Some examples are outlined in Table 9-5.

A lateral chest radiograph is used in conjunction with the PA radiograph to verify the location of any abnormal findings in the lungs. Using Figure 9-8 for example, lesions in the LUL lingula segments (#5) and those in the LLL superior segment (#11) would be indistinguishable with the PA radiograph alone. Using a lateral projection in conjunction with the PA projection, the exact location (#5 or #11) can be verified.

✓ A lateral chest radiograph is used in conjunction with the PA radiograph to verify the location of any abnormal findings in the lungs.

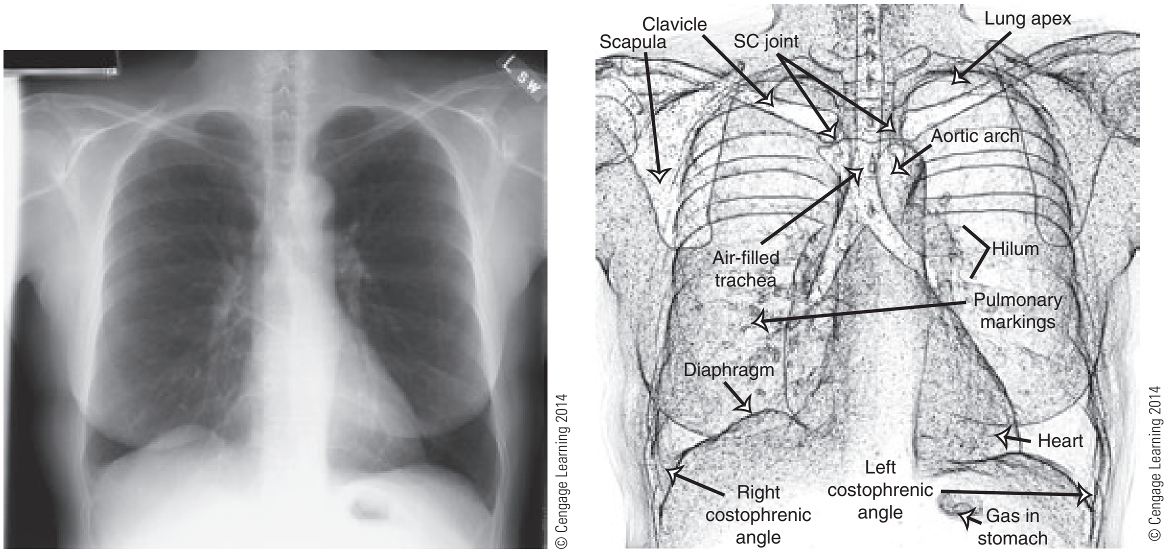

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FIGURE 9-9 A normal posterior-anterior (PA) chest radiograph and corresponding anatomical structures.

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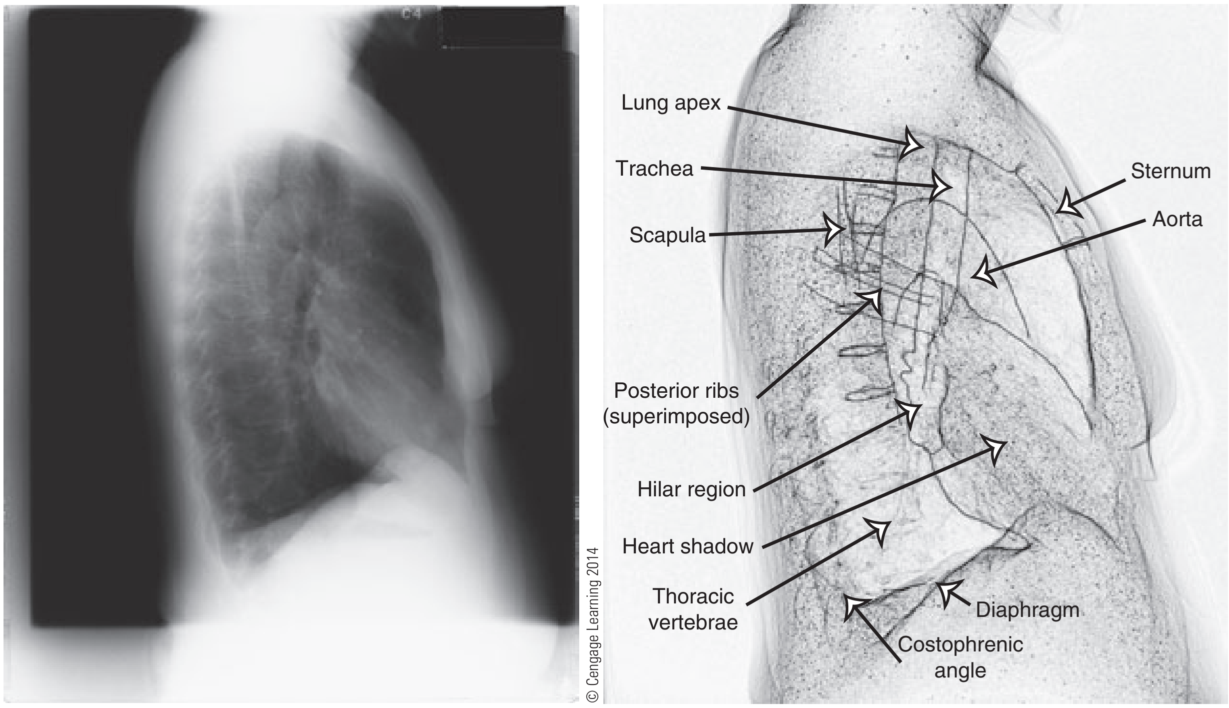

FIGURE 9-10 A normal lateral chest radiograph and corresponding anatomical structures.

TABLE 9-5 Normal and Abnormal Chest Radiograph Appearance

Normal Chest Radiograph Appearance	Abnormal Appearance and Probable Cause
1. Midline trachea and mediasternum	Shift of trachea or mediasternum may occur due to patient rotation during taking of X-ray. Shift of trachea or mediasternum to affected side (e.g., atelectasis, pulmonary fibrosis) or to the unaffected side (e.g., tension pneumonthorax)
2. Dark lung parenchyma with mild scattered white shadows (normal lung tissues and small vessels)	Infiltrates (large amount of white shadows) suggest accumulation of secretions, atelectasis.
3. Sharply pointed right and left costophrenic angels	Blunted costophrenic angle suggests accumulation of fluid in pleural space (e.g., pleural effusion, hemothorax, empyema)
4. Smooth and continuing bony structures	Jagged appearance suggests fracture or broken bones.
5. Cardiac shadow along the diaphragm line is < 50% of the distance between the right and left costophrenic angles.	Congestive heart may cause the cardiac shadow to exceed 50% of the distance between the right and left costophrenic angels.

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FLUID BALANCE AND ANION GAP

Mechanical ventilation may affect a patient's renal function and fluid balance. Since fluid balance and electrolyte concentration are related, the **anion gap** may also be affected as a result of positive pressure ventilation. Proper fluid and electrolyte maintenance should be an integral part of mechanical ventilation to prevent these adverse outcomes.

anion gap: The difference between cations (positive ions) and anions (negative ions) in the plasma. The normal range is 15 to 20 when K^+ is included in the calculation (10 to 14 mEq/L when K^+ is excluded).

Fluid Balance

Positive pressure ventilation reduces cardiac output and thus renal perfusion. Urine output is decreased due to hypoperfusion of the kidneys. Mechanical ventilation also reduces urine output as a result of an increase in antidiuretic hormone (ADH) and a reduction of atrial natriuretic factor (ANF). The end result of these changes is decreased fluid output and fluid retention.

✓ Oliguria may be seen after bleeding, diarrhea, renal failure, shock, drug poisoning, deep coma, or hypertrophy of the prostate.

For these reasons, the fluid level of a ventilator patient must be monitored closely because positive pressure ventilation affects fluid balance (intake and output). Fluid intake is recorded by adding all fluids received by the patient to include fluids provided via the intravenous, oral, and nasogastric routes. Fluid output is commonly done by measuring the urine output. **Oliguria** indicates fluid deficiency and may occur as a result of decreased renal perfusion, decreased fluid intake, and decreased cardiac output. Normal urine output is 50 to 60 mL/hour. Urine output of below 20 mL/hour (or 400 mL in a 24-hour period or 160 mL in 8 hours) is indicative of fluid deficiency (Kraus et al., 1993).

oliguria: Below normal urine output.

✓ Urine output of below 20 mL/hour (or 400 mL in a 24-hour period or 160 mL in 8 hours) is indicative of fluid deficiency.

✓ Normal urine output is 50 to 60 mL/hour.

Reduction in cardiac output can be directly attributed to decreased venous return secondary to positive pressure ventilation and increased intrathoracic pressure. Positive pressure ventilation also causes an increase in the production of antidiuretic hormone (ADH) which further reduces the urine output.

Anion Gap

Table 9-6 shows a typical set of electrolyte parameters with their normal results. Using some of these parameters, the anion gap may be calculated and used to assess a patient's overall electrolyte balance. Anion gap is the relationship of the cations (sodium [Na^+] and potassium [K^+]) to the anions (chloride [Cl^-] and bicarbonate [HCO_3^-]). The anion gap may be determined as follows:

$$\text{Anion gap} = Na^+ - Cl^- - HCO_3^-$$

Normal range: 10 to 14 mEq/L
or

$$\text{Anion gap} = Na^+ + K^+ - Cl^- - HCO_3^-$$

Normal range: 15 to 20 mEq/L

✓ See Appendix 1 for example.

TABLE 9-6 Normal Serum Electrolytes

Cation	Concentration (mEq/L)	Anion	Concentration (mEq/L)
Na^+	140 (138 to 142)	Cl^-	103 (101 to 105)
K^+	4 (3 to 5)	HCO_3^-	25 (23 to 27)
Ca^{++}	5 (4.5 to 5.5)	Protein	16 (14 to 18)
Mg^{++}	2 (1.5 to 2.5)	HPO_4^-, $H_2PO_4^-$	2 (1.5 to 2.5)
		SO_4^-	1 (0.8 to 1.2)
		Organic acids	4 (3.5 to 4.5)
Total cations	151	Total anions	151

✓ Metabolic acidosis in the presence of a normal anion gap is usually caused by a loss of base.

✓ Metabolic acidosis in the presence of an increased anion gap is usually due to increased fixed acids.

Metabolic Acidosis and Anion Gap. Metabolic acidosis in the presence of a normal anion gap is usually caused by a loss of base. This condition is called hyperchloremic metabolic acidosis because it is usually related to excessive chloride ions in the plasma.

Metabolic acidosis in the presence of an increased anion gap is usually due to increased fixed acids. These acids may be produced biologically (e.g., renal failure, diabetic ketoacidosis, lactic acidosis), or they may be added from an external source (e.g., poisoning by salicylates and alcohol) (Chang, 2012).

Respiratory Compensation for Metabolic Acidosis. In mechanically ventilated patients with metabolic acidosis, hyperventilation (↓$PaCO_2$) may occur as a compensatory mechanism for metabolic acidosis. One should not assume that respiratory insufficiency (primary alveolar hyperventilation) is present. The cause of metabolic acidosis must be identified and corrected. More importantly, the ventilator frequency must not be reduced due to an abnormally low $PaCO_2$. Otherwise, persistent hyperventilation and worsening of the work of breathing will continue due to a sudden decrease of ventilator frequency (Rooth, 1974).

✓ Severe K^+ depletion can lead to metabolic alkalosis and compensatory hypoventilation.

Metabolic Alkalosis. It is also important to monitor a patient's potassium level during mechanical ventilation. Severe K^+ depletion can lead to metabolic alkalosis and compensatory hypoventilation (Adams et al., 1982). This may prolong the weaning process when mechanical ventilation is needed to supplement the decreasing spontaneous ventilation.

ARTERIAL BLOOD GASES

Arterial blood gas (ABG) analysis provides useful information about a patient's ventilation ($PaCO_2$), oxygenation (PaO_2), and acid-base (pH) status. It is therefore an essential monitoring tool for patients receiving mechanical ventilation as these patients often have gas exchange and acid-base abnormalities. Table 9-7 shows the normal ABG

TABLE 9-7 Blood Gas Parameters and Normal Range for Adults

Parameter	Monitoring	Normal
$PaCO_2$	Ventilatory status	35 to 45 mm Hg
PaO_2	Oxygenation status	80 to 100 mm Hg
pH	Acid-base status	7.35 to 7.45

values for adult patients. See Chapter 12 "Management of Mechanical Ventilation" for examples of abnormal blood gas reports that are caused by external factors.

Assessment of Ventilatory Status

Direct measurement of arterial carbon dioxide tension ($PaCO_2$) via arterial puncture or indwelling catheter is the most accurate method of assessing a patient's ventilatory status. Hypoventilation and respiratory acidosis are present when the $PaCO_2$ is increased with a concurrent decrease in pH. This condition may be corrected by increasing the frequency or tidal volume on the ventilator. On the other hand, the frequency or tidal volume should be reduced when hyperventilation and respiratory alkalosis occur.

When the acid-base imbalance is caused by metabolic acidosis or alkalosis, it calls for a different ventilator management strategy. The underlying metabolic problem must be corrected before changing the ventilator settings. Ventilator tidal volume or frequency adjustment should not be made to "correct" metabolic acid-base abnormalities during mechanical ventilation.

Respiratory Fatigue. The mechanically ventilated patient who develops hypercapnic respiratory failure secondary to increased carbon dioxide production ($\dot{V}CO_2$) should be monitored closely. The $\dot{V}CO_2$ may be increased due to a hypermetabolic state. This condition may lead to increased minute ventilation in an attempt to keep up with the increasing CO_2 production. A prolonged increase in the work of breathing may lead to respiratory muscle fatigue and ventilatory failure. It has been documented that excessive work of breathing (minute ventilation in excess of 10 L/min.) is often associated with poor outcomes when trying to wean the patient from mechanical ventilation (Stoller, 1991).

✓ A prolonged increase in the work of breathing may lead to respiratory muscle fatigue and ventilatory failure.

Patients with depressed central respiratory drive, elevated V_D/V_T, diminished compliance, or respiratory muscle weakness may also develop respiratory fatigue as they are unable to maintain an increased minute ventilation over an extended period of time.

Assessment of Oxygenation Status

Changes in the patient's oxygen status are commonly assessed by (1) arterial oxygen tension (PaO_2), (2) alveolar-arterial oxygen tension gradient [$P_{(A-a)}O_2$ or $(A-a)DO_2$], (3) arterial to alveolar oxygen tension ratio (PaO_2/P_AO_2), and (4) PaO_2 to F_IO_2.

A decreased PaO_2, an increased $P_{(A\text{-}a)}O_2$, a decreased PaO_2/P_AO_2, or a decreased PaO_2/F_IO_2 reflects tissue hypoxia. Table 9-8 outlines the guideline for interpretation of a patient's oxygenation status.

diffusion defect: Pathologic condition leading to impaired gas exchange through the alveolar-capillary membrane (e.g., interstitial or pulmonary edema).

In general, a decrease in PaO_2 with concurrent increase in $P_{(A\text{-}a)}O_2$ is indicative of hypoxemia due to **diffusion defect**, V/Q mismatch, or shunt. A decrease in PaO_2 with little or no increase in $P_{(A\text{-}a)}O_2$ is probably due to hypoventilation and this can be confirmed by an elevated $PaCO_2$ (Tobin, 1990).

$P_{(A\text{-}a)}O_2$ is the difference of P_AO_2 and PaO_2. It can be calculated as follows:

$$P_{(A\text{-}a)}O_2 = P_AO_2 - PaO_2$$

PaO_2 is obtained from arterial blood gas analysis and P_AO_2 can be calculated by the simplified alveolar air equation, as follows:

$$P_AO_2 = (P_B - P_{H_2O}) \times F_IO_2 - \frac{(PaCO_2)}{R}$$

where P_B = barometric pressure, P_{H_2O} = water vapor pressure (generally 47 mm Hg at 37°C), and R = respiratory quotient (estimated to be 0.8 and it may be deleted from equation when the F_IO_2 is greater than 60%). P_AO_2 is mainly affected by changes of F_IO_2, $PaCO_2$, and P_B.

TABLE 9-8 Interpretation of Oxygenation Status

Parameters	Criteria	Interpretation
PaO_2	80–100 mm Hg	Normal
	60–79 mm Hg	Mild hypoxemia
	40–59 mm Hg	Moderate hypoxemia
	<40 mm Hg	Severe hypoxemia
PaO_2/F_IO_2	≤300 mm Hg (PCWP < 18 mm Hg)	Acute lung injury (ALI)
	≤200 mm Hg (PCWP < 18 mm Hg)	Acute respiratory distress syndrome (ARDS)
$P_{(A\text{-}a)}O_2$	Room air	Should be less than 4 mm Hg for every 10 years of age, otherwise hypoxemia
	100% O_2	Every 50 mm Hg difference approximates 2% shunt
PaO_2/PAO_2	$F_IO_2 \geq 30\%$	>75% Normal
		<75% Hypoxemia

(Data from Girault et al., 1994; Malley, 1990; Shapiro et al., 1994.)

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✓ Acute hypoventilation causes respiratory acidosis.

Hypoventilation. Acute hypoventilation causes CO_2 retention (increased $PaCO_2$) and respiratory acidosis. Without supplemental oxygen, hypoventilation leads to hypoxemia as well. This type of hypoxemia should not be treated with oxygen alone as the underlying condition can only be corrected by improving the alveolar ventilation.

ventilation/perfusion (V/Q) mismatch: An abnormal distribution of ventilation and pulmonary blood flow. High V/Q is related to deadspace ventilation, whereas low V/Q is associated with intrapulmonary shunting.

Ventilation/Perfusion Mismatch. When PaO_2 is decreased with little or no change in $PaCO_2$, **ventilation/perfusion (V/Q) mismatch** or intrapulmonary shunt should be suspected. Hypoxemia caused by ventilation/perfusion (V/Q) mismatch is characterized by a normal or low $PaCO_2$, and this type of hypoxemia responds well to moderate levels of supplemental oxygen.

intrapulmonary shunting: Pulmonary circulation that does not come in contact with ventilated alveoli.

✓ When the PaO_2 is decreased with little or no change in $PaCO_2$, V/Q mismatch or intrapulmonary shunt should be suspected.

✓ Hypoxemia caused by intrapulmonary shunting does not respond well to high concentrations of oxygen.

✓ PEEP and oxygen are usually required to correct hypoxemia caused by intrapulmonary shunting.

Intrapulmonary Shunting. Hypoxemia caused by **intrapulmonary shunting** does not respond well to high concentrations of oxygen. This is because shunted blood does not come in contact with ventilated (oxygenated) alveoli. The $PaCO_2$ is usually normal or low because the peripheral chemoreceptors respond readily to hypoxemia by increasing the minute ventilation (West, 2011).

Positive end-expiratory pressure (PEEP) in conjunction with oxygen are usually required to correct hypoxemia caused by intrapulmonary shunting. If hypoventilation is present as documented by an increased $PaCO_2$, ventilatory assistance may also be necessary.

Diffusion Defects. Diffusion abnormalities can cause hypoxemia by three mechanisms: (1) low oxygen pressure gradient, (2) increased alveolar-capillary thickness or diffusion gradient, and (3) decreased alveolar surface area.

A low alveolar-arterial oxygen tension gradient is usually due to reduction in alveolar PO_2. Oxygen therapy increases the alveolar PO_2 and alveolar-arterial PO_2 tension gradient. It is therefore very effective in correcting hypoxemia caused by uncomplicated diffusion defect.

Increased alveolar-capillary thickness or diffusion gradient can be seen in conditions such as pneumonia and pulmonary and interstitial edema. In mild and uncomplicated cases, hypoxemia may be corrected by oxygen therapy.

Decreased alveolar surface area can be seen in emphysema due to destruction of the lung tissues (Tobin, 1990). This type of structural defect is not reversible, but the diffusion problem may be partially managed by oxygen therapy.

Limitations of Blood Gases

Blood gas analysis and monitoring is not without its limitations. Arterial blood sampling is a procedure requiring the puncture of an artery or placement of an arterial catheter. Inaccurate results can occur with introduction of air bubbles, dilution with excessive heparin, or with faulty handling of the sample itself. It is also important to keep in mind that blood gas values reflect an isolated measurement in time rather than a trend. They should be used to correlate and document trends established by noninvasive monitoring devices such as pulse oximetry. Finally, arterial blood gas measurements are generally a late indicator of respiratory failure and of limited use as an early warning sign (Tobin, 1990).

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Nevertheless, arterial blood gases can still provide very useful information when used in conjunction with other monitoring techniques and thorough patient assessment.

OXYGEN SATURATION MONITORING

Oxygen saturation is traditionally measured by arterial blood gases. The arterial oxygen saturation (SaO_2) is not always readily available. A simple and noninvasive method to monitor the oxygen saturation is by using a **pulse oximeter**. The pulse oximetry oxygen saturation (SpO_2) is less accurate than SaO_2 but it can provide quick spot checks or a trend reflecting a patient's oxygenation status.

pulse oximeter: A device that estimates arterial oxygen saturation (SpO_2) by emitting dual wavelengths of light through a pulsating vascular bed.

Pulse Oximetry

Pulse oximetry has become perhaps the most frequently used method of assessing a patient's oxygenation status. This noninvasive method measures the approximate oxyhemoglobin saturation (SpO_2). It is non invasive and easy to use. Pulse oximeters may be used intermittently to "spot check" the SpO_2 or continuously to monitor the patient's SpO_2 trend. Figure 9-11 shows two portable pulse oximeters.

✓ The SpO_2 reading should not be used and reported if a concurrent low perfusion alarm is present.

The pulse oximeter works by emitting dual wavelengths of light through a pulsating vascular field. Proper placement of the probe is necessary to obtain an accurate reading. The heart rate is also measured as the oximeter evaluates each arterial pulse. If the heart rate on the oximeter varies significantly from the actual pulse as measured by palpation or cardiac monitor, it could mean low perfusion state or motion artifact. The SpO_2 reading should not be used and reported if a concurrent low perfusion alarm is present. A good match of actual and oximetry heart rates does not necessarily indicate an accurate SpO_2 reading. The accuracy of the SpO_2 reading should be correlated with the patient's clinical presentation or verified by periodic arterial blood gases.

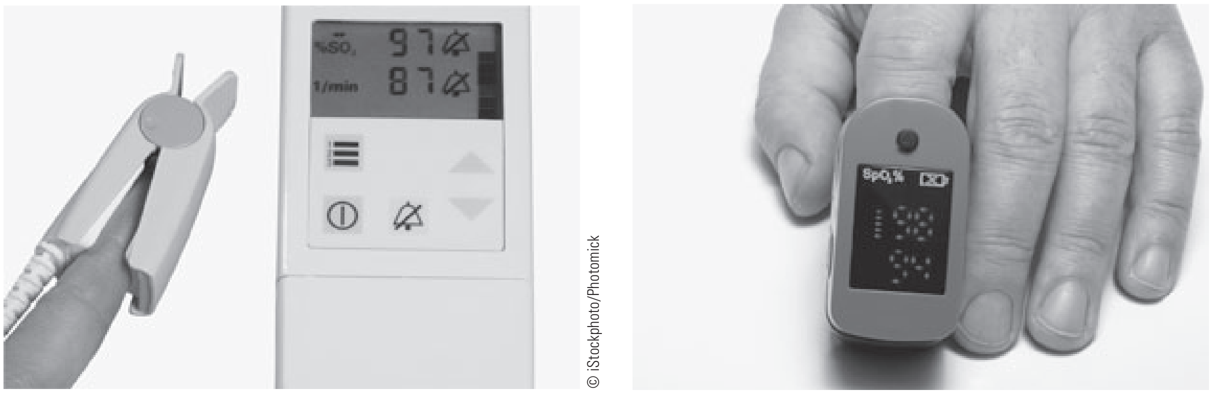

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FIGURE 9-11 Portable pulse oximeters.

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Accuracy and Clinical Use of Pulse Oximetry

✓ SpO_2 above 92% correlates with a PaO_2 above 60 mm Hg.

Pulse oximetry has been used as a reliable noninvasive means of monitoring oxygenation in mechanically ventilated patient. SpO_2 of >95% has a strong correlation with PaO_2 of >70 mm Hg with a sensitivity of 100% (Niehoff et al., 1988).

SpO_2 can be used to facilitate F_IO_2 weaning. The F_IO_2 may be reduced to an appropriate level by use of a single arterial blood gas measurement followed by multiple pulse oximetry measurements (Rotello et al., 1992). Oxygenation of the ventilator-dependent patient can be assured when the SpO_2 is kept above 92% as this level correlates with a PaO_2 above 60 mm Hg (Jubran et al., 1990). Table 9-9 outlines other clinical application of pulse oximetry.

Limitations of Pulse Oximetry

✓ SpO_2 has good correlation with arterial oxygen saturation (SaO_2) when the SaO_2 is 95% or greater.

SpO_2 has good correlation with arterial oxygen saturation (SaO_2) when the SaO_2 is 95% or greater (Niehoff et al., 1988). SpO_2 becomes less accurate as SaO_2 decreases, and over estimation of a patient's oxygenation status may result.

The accuracy of pulse oximetry can be affected by factors such as artifact and underlying patient conditions. Artifact due to motion remains a cause of inaccurate measurement despite corrective efforts (Pologe, 1987). Sunlight has been reported to give a falsely low SpO_2 measurement (Abbott, 1986). Nail polish (primarily blue, green, and black), and intravascular dyes can also give a falsely low SpO_2 reading (Welch, DeCesare & Hess, 1990). Improper placement of the oximeter probe can give a faulty SpO_2 reading as well. If a patient is wearing nail polish, the probe may be placed sideways (White et al., 1989).

dyshemoglobins: Hemoglobins that do not carry oxygen (e.g., carboxyhemoglobin, methemoglobin). In the presence of dyshemoglobins, pulse oximeter reads higher than actual SaO_2.

Pathologic factors such as low perfusion states and presence of **dyshemoglobins** may lead to SpO_2 measurements that are higher than the actual SaO_2 (Schnapp & Cohen, 1990). Table 9-10 shows the factors that affect the accuracy of pulse oximetry.

Integrated Pulse CO-Oximetry

✓ Low perfusion and presence of dyshemoglobins may lead to SpO_2 that are higher than the actual SaO_2.

In addition to the SpO_2 and PR (pulse rate), an integrated pulse CO-oximetry (Masimo Rainbow SET®) uses signal extraction technology to measure a patient's hemoglobin

TABLE 9-9 Clinical Application of Pulse Oximetry

Clinical Application	Examples
Monitor oxygenation status	Mechanical ventilation Intubation Surgery
Titrate F_IO_2	Increase F_IO_2 in hypoxemia Decrease F_IO_2 in weaning
Verify ABG accuracy	Compare O_2 saturation readings to rule out venous sample.

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TABLE 9-10 Factors That Affect the Accuracy of Pulse Oximetry

Factors	Type of Inaccuracy
Sunlight	SpO_2 measures lower than actual SaO_2.
Nail polish	
Fluorescent light	
Intravenous dyes	
Dyshemoglobins (methemoglobin, sulfahemoglobin, carboxyhemoglobin)	SpO_2 measures higher than actual SaO_2.
Low perfusion states	

(SpHb™), oxygen content (SpOC™), carboxyhemoglobin (SpCO®), methemoglobin (SpMet®), pleth variability index (PVI®), and perfusion index (PI).

Pleth variability index (PVI®)(Masimo Corp., Irvine, CA, USA) is an algorithm allowing for automated and continuous calculation of the respiratory variations in the pulse oximeter plethysmographic (ΔPOP) waveform amplitude. PVI has been used to predict fluid responsiveness in mechanically ventilated patients during general anesthesia (Cannesson et al., 2008).

Perfusion index (PI) is a relative assessment of the pulse strength at the monitoring site. The PI display ranges from 0.02% (very weak pulse strength) to 20% (very strong pulse strength). During sensor placement, use a site with the highest PI number (strongest pulse amplitude). The PI is influenced primarily by the amount of blood at the monitoring site, not by the level of oxygenation in the blood. Preliminary data from one study show that low PI values correspond with illness on neonates (DeFelice et al., 2002). Other applications of PI include assessment of pain in the anesthetized state (Hagar et al., 2004) and as an early indicator of successful epidural block in laboring women (Kakazu et al., 2005).

✓ Integrated pulse CO-oximetry is capable of measuring the hemoglobin (SpHb™), oxygen content (SpOC™),carboxyhemoglobin (SpCO®), methemoglobin (SpMet®), pleth variability index (PVI®), and perfusion index (PI).

END-TIDAL CARBON DIOXIDE MONITORING

End-tidal carbon dioxide monitoring is done to monitor a patient's ventilatory status. Once a good correlation is established between $PaCO_2$ and end-tidal PCO_2 ($PetCO_2$), the number of routine blood gases may be reduced. In addition, changes of the $PetCO_2$ values and waveforms may also be obtained and interpreted for additional information about the patient/ventilator system.

end-tidal carbon dioxide monitoring: The CO_2 level measured at the end of exhalation; measured in mm Hg.

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Capnography

Capnography is a measurement of the partial pressure of carbon dioxide in a gas sample. When the sample is collected at the end of expiration, it is called end-tidal partial pressure of carbon dioxide ($PetCO_2$). $PetCO_2$ monitoring provides real-time, noninvasive analysis of a patient's expired CO_2 trend during mechanical ventilation. Ventilators have built-in end-tidal CO_2 monitoring capabilities.

The exhaled CO_2 from the patient ventilator circuit is collected and measured by the infrared absorption technique (Hess, 1990). A mainstream sensor is placed directly onto the ventilator circuit, usually attached to an adaptor on the endotracheal tube. A sidestream sensor aspirates a sample of gas via a small tube connected to the endotracheal tube adaptor. Figure 9-12 illustrates the mainstream and sidestream capnography sensors.

The major advantage of mainstream analysis is the fast response time between actual CO_2 sampling and the display update. The disadvantage of the mainstream adaptor is its excessive weight on the endotracheal tube as well as the additional deadspace in the ventilator/patient circuit. With mainstream sampling, water condensation does not affect analysis; however, secretion buildup on the cell windows can affect the accuracy. A mainstream analyzer also tends to be more frequently handled than the sidestream sensor because the clinician must disconnect it manually to suction the patient (Shelley, 1989).

A sidestream analyzer (aspirating analyzer) places the analyzing mechanism safely within the monitor and draws a sample via a tube connected at the patient's airway (e.g., endotracheal tube). The major advantage with sidestream analysis is the ease of handling, and the analyzer can be attached to other patient devices (e.g., cannula, mask). The major disadvantage is that with periodic aspiration of air samples, secretions and water can be drawn into the sampling tube and cause an occlusion. Lag time for CO_2 display is slightly longer (a few tenths of a second) than the mainstream analyzer but it is negligible. Equipment contamination may be a problem with the sidestream analyzer (Shelley, 1989).

Capnography Waveforms and Clinical Application

A capnogram (Figure 9-13) shows the changes in P_ECO_2 during a complete respiratory cycle. The P_ECO_2 is at zero before exhalation. At the beginning of exhalation, the P_ECO_2 remains at zero as anatomic deadspace volume exits the airways (phase I). The P_ECO_2 then increases dramatically as alveolar gas begins mixing with deadspace gas (phase II). Then the curve plateaus, reflecting the exhalation of alveolar gas (phase III). The end of the "alveolar plateau" is called the end-tidal PCO_2 ($PetCO_2$). Since the $PetCO_2$ approximates the alveolar PCO_2, this value may be used to estimate the $PaCO_2$.

✓ The $PetCO_2$ may be used to estimate the $PaCO_2$.

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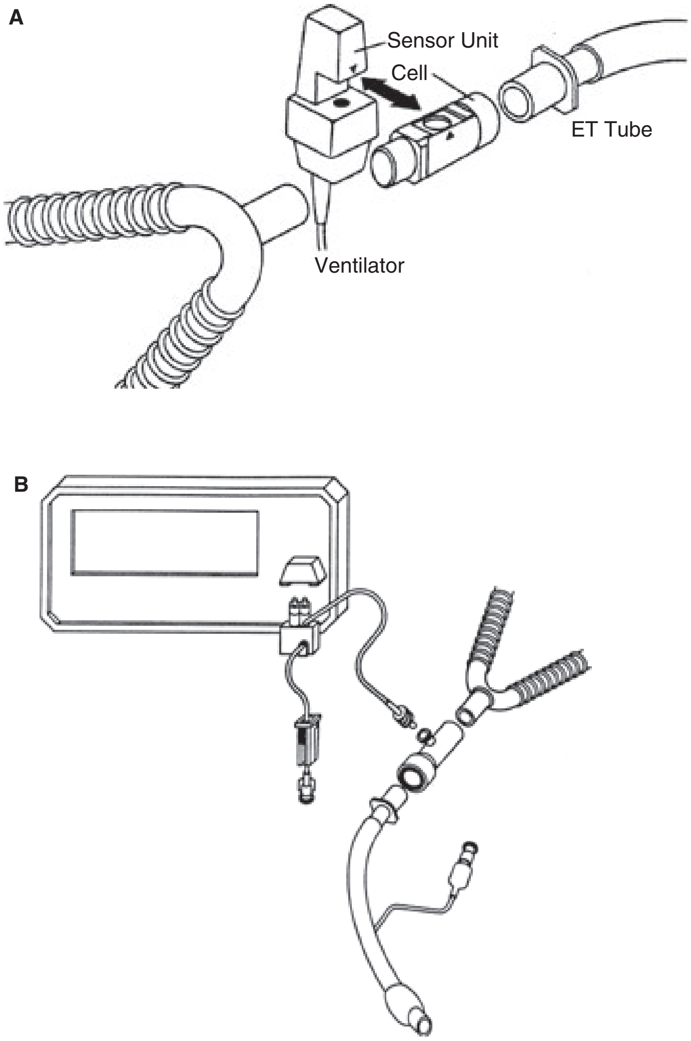

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FIGURE 9-12 (A) Mainstream capnography sensor; (B) Sidestream capnography sensor.

Transitory events can be examined by review of the capnographic tracing. The capnogram can be useful in determining accidental esophageal intubations, endotracheal tube cuff leaks, and airway obstructions. It can also be used to determine the synchronization of respiratory frequencies between the patient and ventilator. Some other clinical applications for capnography include use during weaning, cardiopulmonary resuscitation, intubation, bronchoscopy, and hypocapnic management of patients with head trauma (Carlon et al., 1988; Hess, 1990). The capnographs do not provide absolute measurements but they can be used to follow the PCO_2 changes in hemodynamically stable trauma patients (Hess, 1990). Figure 9-14 shows the representative capnograms that correlate with some clinical conditions.

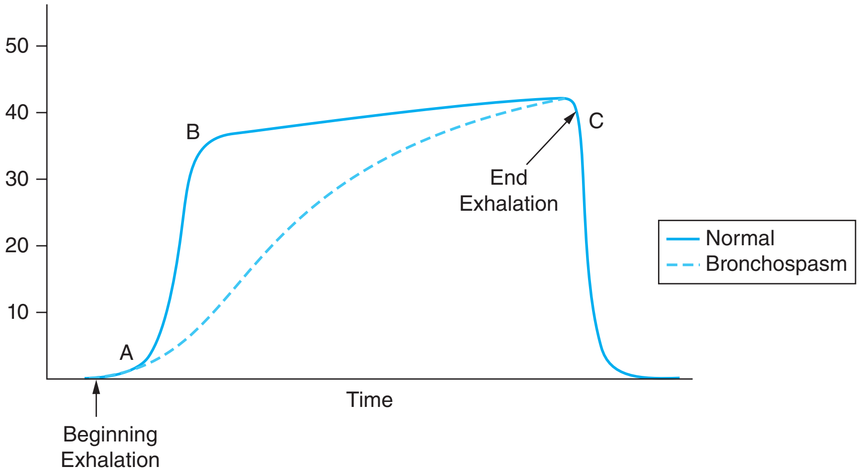

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FIGURE 9-13 The normal capnogram (solid line). (A, phase I) PCO_2 is near zero and it represents gas in anatomic deadspace; (A to B, phase II) PCO_2 rises rapidly as alveolar gas mixes with deadspace gas; (B to C, phase III) alveolar plateau shows arrival of gas flow from alveoli; Point (C) is the end-tidal PCO_2 ($PetCO_2$). Dotted line represents tracing in bronchospasm. (Reference: Hess, 1990.)

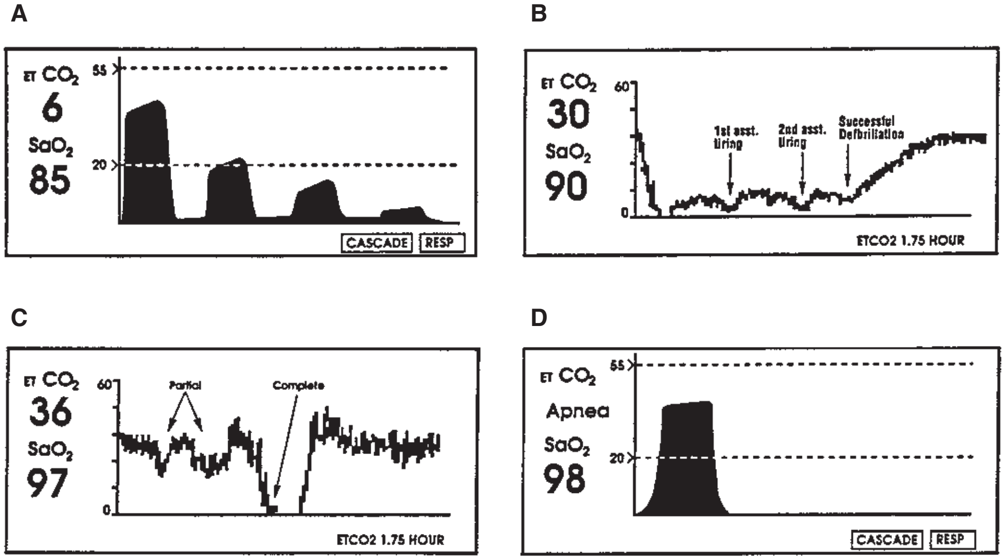

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FIGURE 9-14 Abnormal capnograms monitored via real time or trend screen. (A) Cardiac arrest (real time). The $PetCO_2$ values drop suddenly and proportionally. This condition indicates lung perfusion is inadequate due to a decreased cardiac output. (B) Effectiveness of external compressions can be monitored by the resultant rise in $PetCO_2$ as shown in the trend screen. (C) Kinked ET tube (trend screen). As the ET tube is obstructed (partially or completely), the $PetCO_2$ values reflect the degree of obstruction. With complete obstruction, zero $PetCO_2$ is seen. (D) Disconnection. Immediate disappearance of the $PetCO_2$ values and wave form are observed.

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P(a-et)CO_2 Gradient

✓ Under ideal conditions, the $PaCO_2$ is about 2 mm Hg higher than the $PetCO_2$ resulting a P(a-et)CO_2 of 2 mm Hg.

The correlation between $PaCO_2$ and $PetCO_2$ is excellent and the P(a-et)CO_2 gradient (difference) between these two measurements is about 2 mm Hg in normal individuals. For critical patients, a gradient of 5 mm Hg is considered acceptable (Niehoff et al., 1988).

The P(a-et)CO_2 gradient is primarily affected by alveolar deadspace ventilation (Perel & Stock, 1992), old age, presence of pulmonary disease, and changes in mechanical volume and modality. Table 9-11 lists some specific conditions that increase the P(a-et)CO_2 gradient.

Disposable $ETCO_2$ Detector. Capnography can also be estimated via a low-cost, disposable, plastic, CO_2 (pH)-sensitive device. With this device attached to the endotracheal tube, one can quickly differentiate tracheal from esophageal intubation (Hess, 1990). This occurs when the pH-sensitive device on the material senses the changes in CO_2 concentration.

Limitations of Capnography Monitoring

✓ Capnography readings reflect only the changes in a patient's ventilatory status, rather than the improvement or deterioration of the patient.

✓ Decreased $PetCO_2$ may not be indicative of improvement in gas exchange.

Capnography readings reflect only the changes in a patient's ventilatory status, rather than the improvement or deterioration of the patient (Whitaker, 2001). An example of this is deadspace ventilation as seen in pulmonary embolism (Figure 9-15). A decrease in $PetCO_2$ due to physiologic deadspace ventilation does not mean that the patient's ventilatory status has improved. Lowering the ventilator frequency in this situation could lead to grave consequences.

Other conditions leading to an increase in deadspace ventilation (thus a decrease in $PetCO_2$) are hypotension and high intrathoracic pressure secondary to mechanical ventilation (Whitaker, 2001). This could cause the practitioner to incorrectly assume that the decreased $PetCO_2$ indicates an improvement in gas exchange.

TABLE 9-11 Factors That Increase the P(a-et)CO_2 Gradient

Clinical Condition	Factors
Ventilation	Increased deadspace ventilation Positive pressure ventilation
Perfusion	Decreased cardiac output Decreased pulmonary perfusion Cardiac arrest Pulmonary embolic disease
Temperature	Hyperthermia Hypothermia

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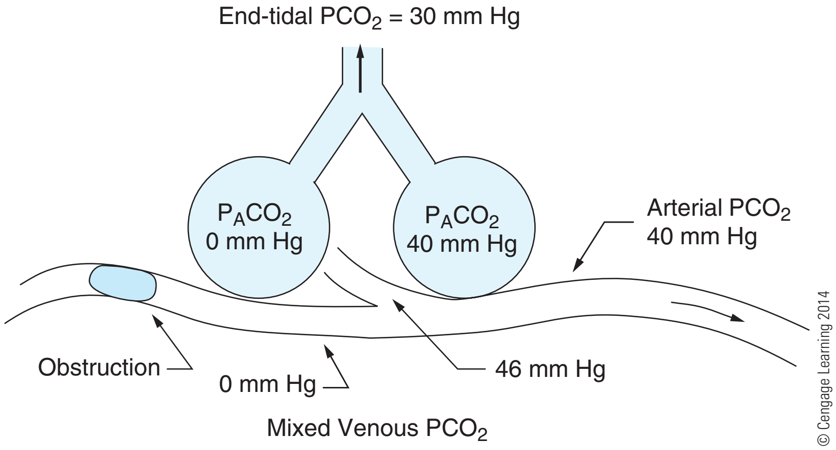

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FIGURE 9-15 Deadspace ventilation induced by blockage of a portion of the pulmonary blood flow (e.g., pulmonary embolism). This condition leads to a reduced $PetCO_2$ reading.

TRANSCUTANEOUS BLOOD GAS MONITORING

Transcutaneous blood gas monitoring involves placement of a miniature Clark (PO_2) or a Severinghaus (PCO_2) electrode on the skin via a double-sided adhesive disk. A heating coil in the electrode increases the permeability of the epidermis, thus facilitating diffusion of gas from the underlying capillaries to the electrode. Transcutaneous blood gas monitoring has been used more often in neonates than in adults (Eberhard et al., 1981).

Transcutaneous PO_2 ($PtcO_2$)

transcutaneous PO_2 ($PtcO_2$): Measurement of PO_2 through the skin by means of a miniature Clark (PO_2) electrode.

For an adequate blood flow to the skin, a heating element is used to provide a constant temperature (e.g., 44°C) to the skin.

The clinical optimal range of $PtcO_2$ for most infants is 50 mm Hg to 70 mm Hg.

The **transcutaneous PO_2 ($PtcO_2$)** provides a noninvasive measurement of arterial oxygen tension. The $PtcO_2$ monitor uses a combined platinum and silver electrode covered by an oxygen-permeable hydrophobic (water-repelling) membrane, with a built-in reservoir of phosphate buffer and potassium chloride. Since the $PtcO_2$ sensor requires an adequate blood flow to the skin, a small heating element is placed in the silver anode to provide a constant temperature (e.g., 44°C) to the skin. Following the initial setup or site change, the $PtcO_2$ should be correlated with an arterial or capillary sample. The value from the $PtcO_2$ monitor should be recorded immediately after obtaining the arterial or capillary sample (Klein, 2008).

In neonates, the transcutaneous PO_2 ($PtcO_2$) closely approximates the PaO_2. But in adults, the $PtcO_2$ measures lower than the actual PO_2 due to thicker skin in adults. For this reason, pulse oximetry (SpO_2) is the preferred method to monitor the oxygenation status of adult patients.

$PtcO_2$ also approximates the central organ PO_2 (Tremper et al., 1979). It has a good correlation with the cardiac output changes in a mechanically ventilated patient (Shapiro et al., 1989). Since $PtcO_2$ values correlate well with arterial values within the whole PO_2 range (particularly in the PO_2 range below 100 mm Hg),

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the $PtcO_2$ can be used as an indicator of hypoxemia (Huch et al., 1974). The clinical optimal range of $PtcO_2$ for most infants is 50 mm Hg to 70 mm Hg (Klein, 2008).

✓ Accuracy of the $PtcO_2$ electrode is affected by skin edema, hypothermia, and capillary perfusion status.

Limitations. Accuracy of the $PtcO_2$ electrode is affected by skin edema, hypothermia, and capillary perfusion status. $PtcO_2$ becomes less accurate when the measuring range is greater than 80 mm Hg (Palmisano et al., 1990). When cardiac output decreases, as with the patient in shock, a disproportionate fall in $PtcO_2$ occurs.

Two other disadvantages of transcutaneous monitors are the need for frequent site changes (every 4 hours) to avoid erythemia and burns to the infant's skin, and a long equilibration time after each site change.

Transcutaneous PCO_2 ($PtcCO_2$)

transcutaneous PCO_2 ($PtcCO_2$): Measurement of PCO_2 through the skin by means of a miniature Severinghaus (PCO_2) electrode.

Transcutaneous PCO_2 ($PtCO_2$) monitoring is done to provide a means of continuous ventilatory assessment. The $PtcCO_2$ is measured by heating the underlying skin to 44°C (40°C to 42°C in neonates, maximum 45°C), which facilitates CO_2 diffusion across the skin to the CO_2 electrode.

The correlation between $PtcCO_2$ and $PaCO_2$ is good in neonates as long as perfusion is normal. This correlation in adults shows mixed results, but in general the $PtcCO_2$ may be useful as a monitoring tool once the trend has been established.

Limitations. It should be noted that $PtcCO_2$ values are usually higher than $PaCO_2$ values. This is due to increased CO_2 production as underlying tissues are heated (Marini, 1988). In addition, during shock or low perfusion states, the $PtcCO_2$ measures higher than the actual $PaCO_2$ due to increased accumulation of tissue CO_2 (Tremper et al., 1981).

CEREBRAL PERFUSION PRESSURE

✓ Maintenance of adequate CPP reduces mortality.

cerebral perfusion pressure (CPP): Pressure required to provide blood flow, oxygen, and metabolite to the brain. CPP = MAP − ICP. Normal range = 70 to 80 mm Hg.

✓ CPP should range between 70 and 80 mm Hg.

Cerebral perfusion pressure (CPP) is the pressure required to provide blood flow, oxygen, and metabolites to the brain. Under normal conditions, the brain regulates its own blood flow regardless of the systemic blood pressure and cerebral vascular resistance. This autoregulation may be lost following head trauma, where the cerebral vascular resistance is often greatly elevated. The brain also becomes vulnerable to changing blood pressures. Depending on the degree of decrease in cerebral perfusion, effects on the brain may range from cerebral ischemia to brain death (Bouma et al., 1990; Marion et al., 1991).

The optimum level of CPP is not defined, but the critical threshold is believed to be from 70 to 80 mm Hg. Mortality increases about 20% for each 10 mm Hg drop in CPP. In studies involving severe head injuries, 35% reduction in mortality was achieved when the CPP was maintained above 70 mm Hg (Bouma et al., 1992; Rosner et al., 1990).

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✓ A higher CPP may be maintained by raising the MAP or by lowering the ICP.

CPP is the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP). The relationship is shown as follows:

$$\text{CPP} = \text{MAP} - \text{ICP}$$

Based on the relationship of MAP and ICP, a higher CPP may be maintained by raising the MAP or by lowering the ICP. In clinical practice ICP is usually controlled within normal limits (i.e., <20 mm Hg). However, it is unknown whether ICP control is necessary, providing that CPP is maintained above the critical threshold by raising the MAP (Changaris et al., 1987; Rosner et al., 1990).

✓ The normal ICP is 8 to 12 mm Hg; clinical practice < 20 mm Hg.

In the absence of hemorrhage, the MAP should be managed initially by maintaining an adequate fluid balance. It may then be followed by using a vasopressor such as norepinephrine or dopamine. Systemic hypotension (i.e., SBP <90 mm Hg) should be avoided and controlled as soon as possible because adequate systemic perfusion is necessary to prevent cerebral ischemia (due to lack of cerebral blood flow). For patients with severe brain injury, systemic hypotension contributes to an increased morbidity and mortality. (Chesnut et al., 1993; Marmarou et al., 1991).

✓ Systemic hypotension is associated with poor outcomes in patients with severe head injuries.

SUMMARY

Monitoring in mechanical ventilation is done to provide information about the condition of the patient and the overall effectiveness of a treatment plan. The results obtained from different monitoring techniques should be used and interpreted together and should not be treated as isolated measurements. For example, a decrease in end-tidal CO_2 may indicate the presence of deadspace ventilation. But this assumption must be confirmed with other supporting evidence such as a concurrent decrease in perfusion (decrease in cardiac output or other hemodynamic values). Trending or interpreting a series of measurements is also more meaningful since the overall condition of a patient is a dynamic process, not a set of separated events.

Finally, the condition of the patient should be assessed in conjunction with the monitoring results. This is because the patient may temporarily compensate for abnormal conditions under extremely stressful settings. This erroneous "normal" measurement may not be apparent by reviewing the laboratory results alone. Therefore, careful examination of the patient should always be a vital part of monitoring in mechanical ventilation.

Self-Assessment Questions

1. A patient suddenly develops shortness of breath and the SpO_2 drops to 87%. The therapist should anticipate a moderate increase of all of the following measurements *except:*

A. heart rate.
B. minute ventilation.
C. respiratory frequency.
D. temperature.

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2. Prolonged suctioning of the lungs via the endotracheal tube is not advised because this can induce:

   A. hypovolemia and shock.
   B. bradycardia and arrhythmia.
   C. hyperventilation.
   D. productive coughs.

3. One of the complications of positive pressure ventilation is that it can cause a(n) ________ venous return and ________.

   A. increased, hypertension
   B. increased, hypotension
   C. decreased, hypertension
   D. decreased, hypotension

4. A blood gas sample was collected from a hypothermic patient whose core temperature was 24°C. If the sample was analyzed at 37°C with a PO_2 of 90 mm Hg, the temperature corrected PO_2 (to patient's 24°C) would be:

   A. negligible or almost 90 mm Hg.
   B. much higher than 90 mm Hg.
   C. much lower than 90 mm Hg.
   D. dependent on the position of the oxyhemoglobin curve.

5. A physician asks the therapist to evaluate the breath sounds of a patient who has an admitting diagnosis of pneumonia. By placing the stethoscope diaphragm next to the side of the spine below the scapula, the therapist is listening to the breath sounds of the ________ segments of the ________ lobes.

   A. posterior, upper
   B. superior, middle
   C. posterior, middle
   D. superior, lower

6 to 9. Match the breath sounds with the conditions that may be the cause of these abnormalities. Use each answer only once.

Breath Sound	**Condition**
6. Diminished or absent	A. Lung consolidation
7. Wheezes	B. Airway obstruction
8. Inspiratory crackles	C. Excessive secretions
9. Coarse crackles	D. Airway narrowing

10. Excessive positive pressure or volume during mechanical ventilation may ________ the intrathoracic pressure and ________ the cardiac and urine outputs.

   A. increase, increase
   B. increase, decrease
   C. decrease, increase
   D. decrease, decrease

11. Infiltrates appear to be ________ shadows on the chest radiograph and they are caused by presence of ________.

   A. black, blood or fluid
   B. black, secretions or atelectasis
   C. white, blood or fluid
   D. white, secretions or atelectasis

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12. Metabolic acidosis (low bicarbonate) with a(n) ________ anion gap is called hyperchloremic metabolic acidosis because the excessive chloride (Cl^-) ions ________ the deficiency of bicarbonate (HCO_3^-) ions in the plasma.

    A. normal, offset
    B. normal, aggravate
    C. abnormal, offset
    D. abnormal, aggravate

13. A patient who has been undergoing the weaning process from mechanical ventilation suddenly stops breathing spontaneously. The therapist should expect to see the following changes on the next set of blood gas results *except:*

    A. increase in PO_2.
    B. increase in PCO_2.
    C. decrease in pH.
    D. decrease in HCO_3^-.

14. In blood gas interpretation, the $PaCO_2$ is primarily used to assess a patient's ________ status, and the PO_2 is useful for the ________ status.

    A. ventilatory, acid-base
    B. ventilatory, oxygenation
    C. acid-base, oxygenation
    D. acid-base, ventilatory

15 to 17. Match the causes of hypoxemia with the characteristics that may be used to distinguish these abnormalities. Use each answer only once.

Cause of Hypoxemia	**Characteristics**
15. Hypoventilation	A. Normal or low $PaCO_2$; hypoxemia does not respond to high levels of oxygen.
16. V/Q mismatch or diffusion defects	B. High $PaCO_2$; hypoxemia improves with ventilation and low levels of oxygen.
17. Intrapulmonary shunt	C. Normal or low $PaCO_2$; hypoxemia responds very well to moderate levels of oxygen.

18. A patient rescued from a house fire has an admitting diagnosis of severe smoke inhalation. She is breathing spontaneously and receiving 100% oxygen via a non-rebreathing mask. Due to her condition, pulse oximetry ________ be done because ________.

    A. should, of cost savings
    B. should, continuous monitoring is being done
    C. should not, the SpO_2 reading will be higher than actual SaO_2
    D. should not, the SpO_2 reading will be required lower than actual SaO_2

19. In metabolic acidosis, patients with adequate lung function are capable of compensating for this condition by ________. This would cause the end-tidal CO_2 readings to be ________ than normal.

    A. hyperventilation, higher
    B. hyperventilation, lower
    C. hypoventilation, higher
    D. hypoventilation, lower

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20. Which of the following statements is *not* true regarding transcutaneous oxygen and carbon dioxide monitors?

    A. Site changes are required every 3 to 4 hours.
    B. Long equilibration time for calibration.
    C. Accurate PO_2 and PCO_2 measurements.
    D. More accurate in neonatal use.

21. In order to ensure adequate blood flow to the brain, the cerebral perfusion pressure (CPP) should be maintained between:

    A. 0 and 20 mm Hg.
    B. 30 and 50 mm Hg.
    C. 70 and 80 mm Hg.
    D. 100 and 120 mm Hg.

Answers to Self-Assessment Questions

1. D.
2. B.
3. D.
4. C.
5. D.
6. B.
7. D.
8. A.
9. C.
10. B.
11. D.
12. A.
13. A.
14. B.
15. B.
16. C.
17. A.
18. C.
19. B.
20. C.
21. C.

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Chapter 10

Hemodynamic Monitoring

David W. Chang
Gary Hamelin

Outline

- Introduction
- Invasive Hemodynamic Monitoring
  - *Technical Background*
  - *Units of Measurement*
  - *Types of Catheters*
- Arterial Catheter
  - *Insertion of Arterial Catheter*
  - *Normal Arterial Pressure and Mean Arterial Pressure*
  - *Pulse Pressure*
  - *Potential Problems with Arterial Catheter*
- Central Venous Catheter
  - *Insertion of Central Venous Catheter*
  - *Components of Central Venous Pressure Waveform*
  - *CVP Measurements*
- Pulmonary Artery Catheter
  - *Insertion of Pulmonary Artery Catheter*
  - *Components of Pulmonary Arterial Pressure Waveform*
  - *PAP Measurements*
  - *Pulmonary Capillary Wedge Pressure*
  - *Components of Pulmonary Capillary Wedge Pressure Waveform*
  - *PCWP Measurements*
  - *Verification of the Wedged Position*
  - *Cardiac Output and Cardiac Index*
- Summary of Preloads and Afterloads
- Calculated Hemodynamic Values
  - *Stroke Volume and Stroke Volume Index*
  - *Oxygen Consumption and Oxygen Consumption Index*
  - *Pulmonary Vascular Resistance*
  - *Systemic Vascular Resistance*
- Mixed Venous Oxygen Saturation
  - *Decrease in Mixed Venous Oxygen Saturation*
  - *Increase in Mixed Venous Oxygen Saturation*
- Less-Invasive Hemodynamic Monitoring

- *Pulse Contour Analysis*

Noninvasive Hemodynamic Monitoring
- *Transesophageal Echocardiography*
- *Carbon Dioxide Elimination ($\dot{V}CO_2$)*

Impedance Cardiography
- *Theory of Operation*
- *Thermodilution Method and ICG*
- *Accuracy of ICG*
- *Clinical Application*

Summary

Self-Assessment Questions

Answers to Self-Assessment Questions

References

Key Terms

- afterload
- carbon dioxide elimination ($\dot{V}CO_2$)
- cardiac index
- cardiac output
- central venous pressure
- contractility
- hemodynamic monitoring
- impedance cardiography (ICG)
- mean arterial pressure
- preload
- pulmonary vascular resistance (PVR)
- pulse contour analysis
- stroke volume
- systemic vascular resistance
- transesophageal echocardiography
- venous return

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Identify or calculate from an arterial waveform the systolic pressure, diastolic pressure, mean arterial pressure, dicrotic notch, and pulse pressure.
- Describe the proper placement, waveform, and normal values obtained from a central venous catheter.
- Outline the clinical application of central venous pressure measurements.
- Describe the proper placement, waveform, and normal values obtained from a pulmonary artery catheter.
- Outline the clinical application of pulmonary artery pressure and pulmonary capillary wedge pressure.
- Calculate and describe the clinical application of: stroke volume and index, oxygen consumption and index, pulmonary vascular resistance, and systemic vascular resistance.
- Describe the theory of operation and clinical application of pulse contour analysis, transesophageal echocardiography, carbon dioxide elimination, and impedance cardiography.

INTRODUCTION

Evolving technology in hemodynamic monitoring has been a useful adjunct in the management of patients with cardiovascular instability. This monitoring technology was initially developed in the 1970s using invasive methods. In recent years, monitoring technology has undergone substantial changes to include less-invasive and noninvasive techniques. Hemodynamic monitoring is not intended for every patient who requires mechanical ventilation. For many critically ill patients, hemodynamic data can add valuable information to the overall management strategy.

In the most basic sense, hemodynamic monitoring is the measurement of the force (pressure) exerted by the blood in the vessels or heart chambers during systole and diastole.

In addition to systolic and diastolic pressures in both the systemic and pulmonary circulations, hemodynamic monitoring equipment also measures cardiac output and mixed venous oxygen saturation. These and other direct measurements gathered through hemodynamic monitoring can be used to calculate other values for different clinical applications.

INVASIVE HEMODYNAMIC MONITORING

Invasive **hemodynamic monitoring** requires the use of the central venous and pulmonary artery catheters. The central venous catheter measures the **central venous pressure** (right ventricular **preload**), and the pulmonary artery catheter measures the pulmonary artery pressure (right ventricular **afterload**) and the pulmonary capillary wedge pressure (left ventricular preload). Impedance cardiography is a noninvasive method to measure and calculate selected hemodynamic parameters.

hemodynamic monitoring: Measurement of the blood pressure in the vessels or heart chambers during contraction (systole) and relaxation (diastole).

central venous pressure (CVP): Pressure measured in the vena cava or right atrium. It reflects the status of blood volume in the systemic circulation. Right ventricular preload.

preload: The end-diastolic stretch of the muscle fiber.

afterload: The resistance of the blood vessels into which the ventricle is pumping blood.

Technical Background

Measurement of hemodynamic pressures is based on the principle that liquids are noncompressible and that pressures at any given point within a liquid are transmitted equally. When a closed system is filled with liquid, the pressure exerted at one point can be measured accurately at any other point on the same level. For example, if a catheter is placed into the radial artery facing the flow of blood and then connected directly to a tubing that is filled with liquid, the pressure exerted by the blood at the tip of the catheter will be accurately transmitted to the liquid-filled tubing. This pressure signal can then be changed to an electronic signal by a transducer and amplified and displayed on a monitor as both a waveform and digital display.

Hemodynamic monitoring is generally done by using a combination of arterial catheter, central venous catheter, and pulmonary artery catheter. One or more of these catheters are introduced into the blood vessel, advanced to a suitable location, and then connected to a monitor at the patient's bedside. The display on the monitor

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TABLE 10-1 Conversions of mm Hg and kilopascal (kPa)

From mm Hg to kPa	From kPa to mm Hg
mm Hg × 0.133 = kPa	kPa × 7.501 = mm Hg

✓ Invasive hemodynamic monitoring uses a transducer to convert a pressure signal (in the catheter) to an electronic signal (on the monitor).

is made possible by using a transducer and an amplifier between the catheter and monitor. Invasive hemodynamic monitoring uses a transducer to convert a pressure signal (in the catheter) to an electronic signal (on the monitor).

To ensure accurate measurements, the transducer, catheter, and measurement site should all be at the same level. Otherwise, the force of gravity will alter the actual readings. For example, a higher reading may be obtained if the transducer and catheter are located lower than the measurement site.

As with other invasive procedures, hemodynamic monitoring should only be used as indicated because infection, dysrhythmia, bleeding, and trauma to blood vessels are potential complications.

Units of Measurement

Hemodynamic pressure readings are measured in units of millimeters of mercury (mm Hg) in the United States and in kilopascals (kPa) in other countries using Système International (SI) units. The conversion factors in Table 10-1 may be used to change between mm Hg and kPa pressure units. Hemodynamic readings begin with the atmospheric pressure as the zero point. Since changes in atmospheric pressure are gradual and insignificant, adjustments are not necessary in trending measurements.

Types of Catheters

✓ The proximal opening in the pulmonary artery catheter can also measure the right atrial pressure (i.e., CVP).

Three different catheters are used in invasive hemodynamic monitoring: arterial catheter, central venous catheter, and pulmonary artery catheter. The arterial catheter is used to monitor systemic arterial pressure. Central venous pressure is measured by a catheter in the superior vena cava or right atrium. A pulmonary artery catheter (i.e., Swan-Ganz catheter) is used to measure the pulmonary arterial pressure and pulmonary capillary wedge pressure. The proximal opening in the pulmonary artery catheter can also measure the pressure in the right atrium. The insertion sites, location, and uses of hemodynamic catheters are summarized in Table 10-2.

ARTERIAL CATHETER

In hemodynamically unstable patients who are receiving fluid infusion or drugs to improve circulation, continuous and accurate blood pressure measurements are essential. With an arterial catheter, most bedside monitors will display a graphic

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TABLE 10-2 Insertion Sites, Location, and Uses of Hemodynamic Catheters

Catheter	Insertion Sites	Location	Common Uses
Arterial	Radial (first choice), brachial, femoral, or dorsalis pedis artery	Within systemic artery near insertion site	(1) Measure systemic artery pressure. (2) Collect arterial blood gas samples.
Central venous	Subclavian or internal jugular vein	Superior vena cava near right atrium or within right atrium	(1) Measure central venous pressure. (2) Administer fluid or medication.
Pulmonary artery	Subclavian or internal jugular vein	Branch of pulmonary artery	(1) Measure CVP, PAP, and PCWP. (2) Collect mixed venous blood gas samples. (3) Monitor mixed venous O_2 saturation. (4) Measure cardiac output. (5) Provide cardiac pacing.

waveform as well as a digital readout of systolic pressure, diastolic pressure, and **mean arterial pressure.**

mean arterial pressure: The average blood pressure in the arterial circulation. Normal is >60 mm Hg.

✓ Collateral circulation to the hand must be confirmed by the Allen test before radial artery puncture or catheterization.

Insertion of Arterial Catheter

Systemic arterial pressure is measured by placing an arterial catheter into the radial artery. The brachial, femoral, or dorsalis pedis arteries may also be used, but the radial artery remains the first choice because of the availability of collateral circulation to the hand provided by the ulnar artery. The femoral artery is sometimes used to monitor left atrial pressures during cardiac surgery.

Correct placement of the arterial catheter may be assessed by the appearance of an arterial waveform on the monitor (Figure 10-1). Once in place, an arterial line provides continuous, direct measurement of systemic blood pressure as well as convenient access to arterial blood gas samples. Although this invasive procedure has potential complications such as bleeding, blood clot, and infection, it has advantages over noninvasive monitoring of blood pressure. Use of a sphygmomanometer (blood pressure cuff) can be simpler and safer, but inaccuracies may occur in conditions of improper technique, increased vascular tone, and vasoconstriction (Keckeisen, 1991).

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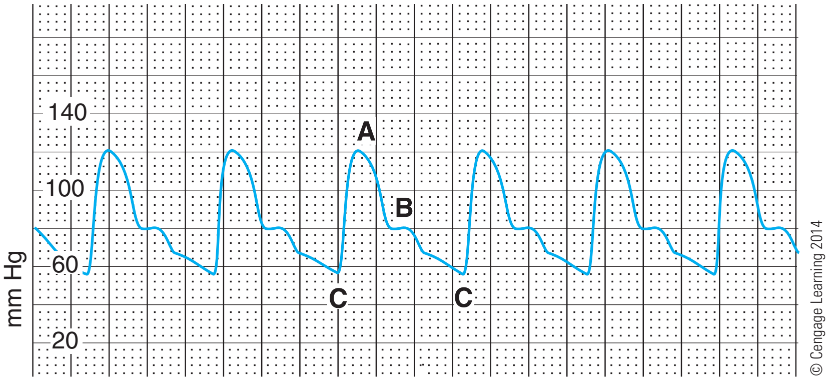

FIGURE 10-1 Normal arterial pressure waveform. The systolic and diastolic pressures are about 120 and 60 mm Hg, respectively. (A) Systolic pressure; (B) Dicrotic notch; (C) End-diastolic pressure.

Figure 10-1 shows a normal arterial pressure waveform. The systolic upstroke (C to A) reflects the rapid increase of arterial pressure in the blood vessel during systole. The downslope or dicrotic limb (A to C) is caused by the declining pressure that occurs during diastole. The dicrotic notch (B) is caused by the closure of the semilunar valves (mainly aortic valve) during diastole. The lowest point (C) of the tracing represents the arterial end-diastolic pressure.

Normal Arterial Pressure and Mean Arterial Pressure

The normal arterial pressure values are in the range of 100–140 mm Hg systolic and 60–90 mm Hg diastolic in most adults. From the systolic and diastolic pressures, the mean arterial pressure may be calculated as follows:

$$\text{MAP} = \frac{(P_{\text{systolic}} + 2 \times P_{\text{diastolic}})}{3}$$

A normal MAP of 60 mm Hg is considered the minimum pressure needed to maintain adequate tissue perfusion (Bustin, 1986). The diastolic value receives greater weight in this formula because the diastolic phase is about twice as long as the systolic phase. Accuracy of blood pressure readings depends on proper setup and calibration of the monitoring system.

stroke volume: Blood volume pumped by the ventricles in one contraction.

Since arterial pressure is the product of **stroke volume** (i.e., blood flow) and vascular resistance, changes in either parameter can affect the arterial pressure. Opposing changes of these two parameters (e.g., increase in stroke volume and decrease in vascular resistance) may present an unchanged arterial pressure or mean arterial pressure. Therefore, interpretation of arterial pressure measurements should take the relationship of these two factors into consideration.

Pulse Pressure

Pulse pressure is the difference between arterial systolic and diastolic pressures. Normal pulse pressure ranges from 30 mm Hg to 40 mm Hg. Since the arterial

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TABLE 10-3 Conditions Leading to High Pulse Pressure

Condition	Example
High stroke volume	Hypervolemia
Noncompliant blood vessel	Arteriosclerosis
Abnormal heart rate	Bradycardia Heart Block

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systolic and diastolic pressures are affected by stroke volume and vascular compliance, pulse pressure can be used to assess the gross changes in stroke volume and blood vessel compliance. High pulse pressure may occur in conditions where the stroke volume is high, blood vessel compliance is low, or heart rate is low. Low pulse pressure may occur in conditions where the stroke volume is low, blood vessel compliance is high, or heart rate is high (Christensen, 1992a, 1992b).

✓ Pulse pressure is the difference between arterial systolic and diastolic pressures (normal 30 mm Hg to 40 mm Hg).

High (Wide) Pulse Pressure. High pulse pressure (>40 mm Hg) can occur with an increasing systolic pressure or a decreasing diastolic pressure. The systolic pressure may be increased when the stroke volume is increased or the blood vessel compliance is decreased. As long as the diastolic pressure does not increase by the same proportion, a high pulse pressure results. Bradycardia may also lead to a higher pulse pressure because a slow heart rate allows the blood volume more time for diastolic runoff and causes a lower diastolic pressure. The conditions that may lead to a high pulse pressure are summarized in Table 10-3.

High pulse pressure may be an important risk factor for heart disease. In elderly patients, a 10 mm Hg rise in pulse pressure increases the risk of major cardiovascular complication and mortality by about 20% (Blacher et al., 2000).

Low (Narrow) Pulse Pressure. By the same mechanism, a decreased stroke volume or an increased blood vessel compliance leads to a corresponding decrease in systolic pressure. A low pulse pressure (<30 mm Hg) is seen as long as the diastolic pressure does not decrease by the same proportion. Tachycardia may also lead to a lower pulse pressure because a high heart rate provides less time for diastolic runoff and causes a higher diastolic pressure. The conditions leading to a low pulse pressure are summarized in Table 10-4.

TABLE 10-4 Conditions Leading to Low Pulse Pressure

Condition	Example
Low stroke volume	Congestive Heart Failure
High compliance blood vessel	Septic Shock
Abnormal heart rate	Tachycardia

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TABLE 10-5 Potential Problems with Arterial Catheter

Factors	Problem
Air bubbles in tubing Loose tubing connections	Dampens the pressure signal
Transducer and catheter placed higher than measurement site	Measurement lower than actual
Transducer and catheter placed lower than measurement site	Measurement higher than actual
Inadequate pressure applied to the flush solution bag	Backup of blood in the tubing
Blood clot at catheter tip, catheter tip blocked by wall of artery	Inaccurate reading, signal interference

Potential Problems with Arterial Catheter

Air bubbles and loose tubing connections can "dampen" the pressure signal. Improper leveling of the transducer and catheter can cause false high or false low readings. Inadequate pressure applied to the heparin solution bag can result in backup of blood in the tubing when the arterial pressure becomes higher than the heparin line pressure. Clotting of blood at the catheter tip or blockage of the catheter tip by the wall of the artery can interfere with the hemodynamic signal. The potential problems that are related to the arterial catheter are shown in Table 10-5.

Most intensive care units have standard procedures in place to minimize such problems. Careful adherence to proper setup and calibration of hemodynamic monitoring equipment are essential.

CENTRAL VENOUS CATHETER

The central venous pressure (CVP) can be monitored through a central venous catheter placed either in the superior vena cava near the right atrium or in the right atrium. The pressure measured in the right atrium is right atrial pressure (RAP) but it is commonly called CVP. The RAP can also be monitored via the proximal port of a pulmonary artery catheter.

CVP measures the filling pressures in the right heart and assesses the systemic fluid status and right heart function.

The primary use CVP in hemodynamic monitoring is to measure the filling pressures in the right heart. The CVP is helpful in assessing fluid status and right heart function. However, it is often late to reflect changes in the left heart. The central venous catheter can also be used to collect "mixed" venous blood samples and for administration of medications and fluids. (Note: A true mixed venous blood sample

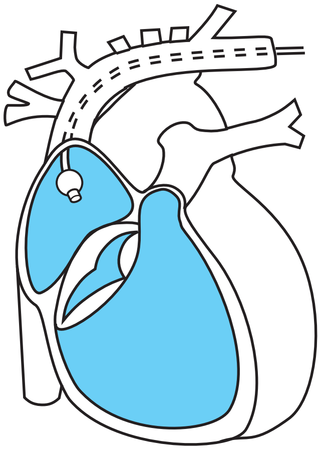

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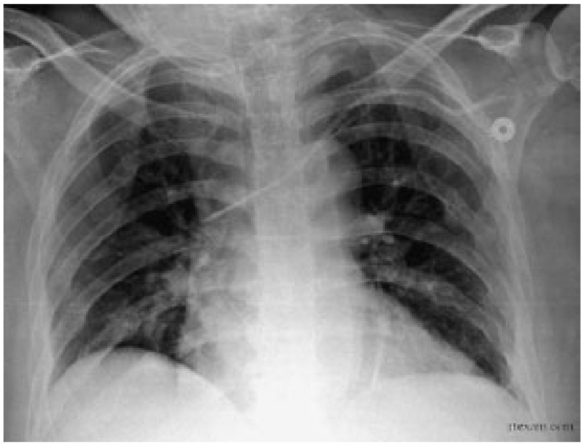
FIGURE 10-2 Position of a central venous (right atrial) catheter.

is obtained from the pulmonary artery via a pulmonary artery catheter.) Figure 10-2 shows the position of the catheter tip of a central venous catheter.

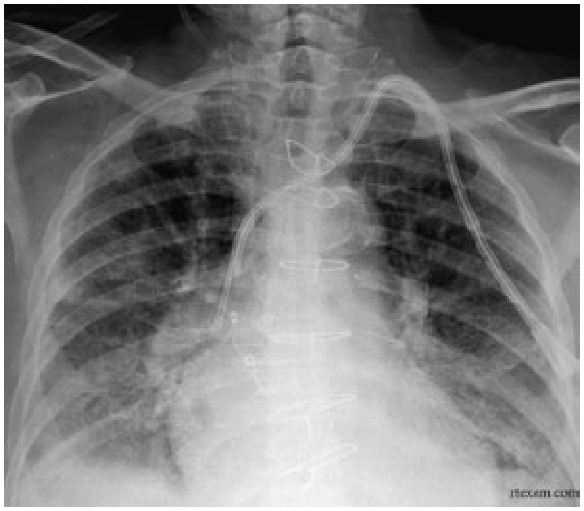
Insertion of Central Venous Catheter

The central venous catheter is commonly inserted through the subclavian vein or the internal jugular vein. Figures 10-3 and 10-4 show the radiographic catheter positions inserted via the left subclavian vein and left internal jugular vein. Continuous monitoring of the central venous pressure should have a typical pressure



Courtesy of rtexam.com

FIGURE 10-3 Left subclavian vein placement of a central venous catheter.



Courtesy of rtexam.com

FIGURE 10-4 Left internal jugular vein placement of a central venous catheter.

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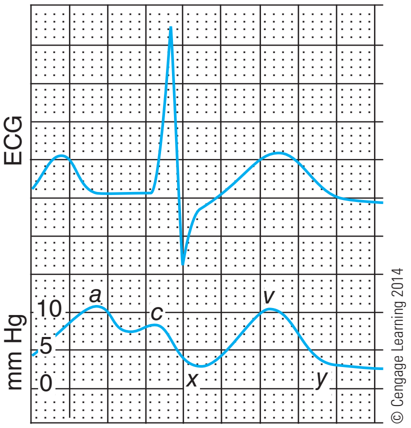

FIGURE 10-5 Tracing of a central venous pressure waveform and the corresponding ECG electrical conduction.

tracing as shown in Figure 10-5. Infection, bleeding, and pneumothorax are potential complications of central venous catheter insertion.

Components of Central Venous Pressure Waveform

Figures 10-5 shows the ECG tracing and the corresponding CVP waveform. Note that the ECG electrical conduction precedes the pressure waveform by a fraction of a second. The upstroke *a* wave reflects right atrial contraction (follows the *p* wave on the ECG), *c* wave reflects closure of the tricuspid valve during systole (appears within the QRS complex on the ECG), *x* downslope occurs as the right atrium relaxes, *v* wave is caused by right ventricular contraction (appears at the *T* wave on the ECG), and *y* downslope reflects ventricular relaxation and rapid filling of blood from the right atrium to the right ventricle.

Abnormal Right Atrial Pressure Waveform. Since each wave or downslope on the right atrial waveform coincides with an event during systole or diastole, changes in the hemodynamic status of the heart will cause changes to certain components of the waveform, particularly the *a* and *v* waves (Schriner, 1989).

The *a* wave on the right atrial waveform may be elevated in conditions in which the resistance to right ventricular filling is increased. Examples include tricuspid valve stenosis, decreased right ventricular compliance due to ischemia or infarction, right ventricular volume overload or failure, pulmonic valve stenosis, and primary pulmonary hypertension. The *a* wave may be absent if atrial activity is absent or extremely weak.

Reflux of blood into the right atrium during contraction due to an incompetent triscupid valve will cause an elevated *v* wave. Elevation of *a* and *v* waves may be seen in conditions such as cardiac tamponade, volume overload, or left ventricular failure.

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TABLE 10-6 Conditions That Affect the Central Venous Pressure

Change	Examples
Decrease in CVP	Absolute hypovolemia (blood loss, dehydration) Relative hypovolemia (shock, vasodilation)
Increase in CVP	Positive pressure ventilation Increased pulmonary vascular resistance Hypervolemia Right ventricular failure Left ventricular failure (late change in CVP)

CVP Measurements

CVP is reported as a mean pressure and its normal range in the vena cava is from 0 to 6 mm Hg. When the measurement is taken in the right atrium, the normal value range is from 2 to 7 mm Hg, slightly higher than the CVP reading (Christensen, 1992a, 1992b).

Since **venous return** is determined by the pressure gradient between the mean arterial pressure and CVP, an increased CVP leads to a smaller pressure gradient and a lower blood return to the right heart. This condition is observed during positive pressure ventilation or as a result of right ventricular failure (e.g., cor pulmonale due to chronic pulmonary hypertension; right-sided myocardial infarction). The conditions that may affect the CVP measurements are summarized in Table 10-6.

venous return: Blood flow from the systemic venous circulation to the right heart.

PULMONARY ARTERY CATHETER

The first pulmonary artery catheter was developed in 1953 and used in dogs by the U.S. physiologists Michael Lategola and Hermann Rahn. In the late 1960s, a more refined pulmonary artery catheter was developed and used in humans by the U.S. physicians Harold James Swan and William Ganz (Swan et al., 1970). The current pulmonary artery catheter (Swan-Ganz catheter) is a flow-directed, balloon-tipped catheter. The addition of thermistor (for **cardiac output** measurement), and light-reflective fiberoptic element (for mixed venous oxygen saturation measurement) to the catheter greatly expanded the scope and capability of hemodynamic monitoring.

cardiac output: Blood volume pumped by the heart in 1 min. Normal range is 4–8 L/min.

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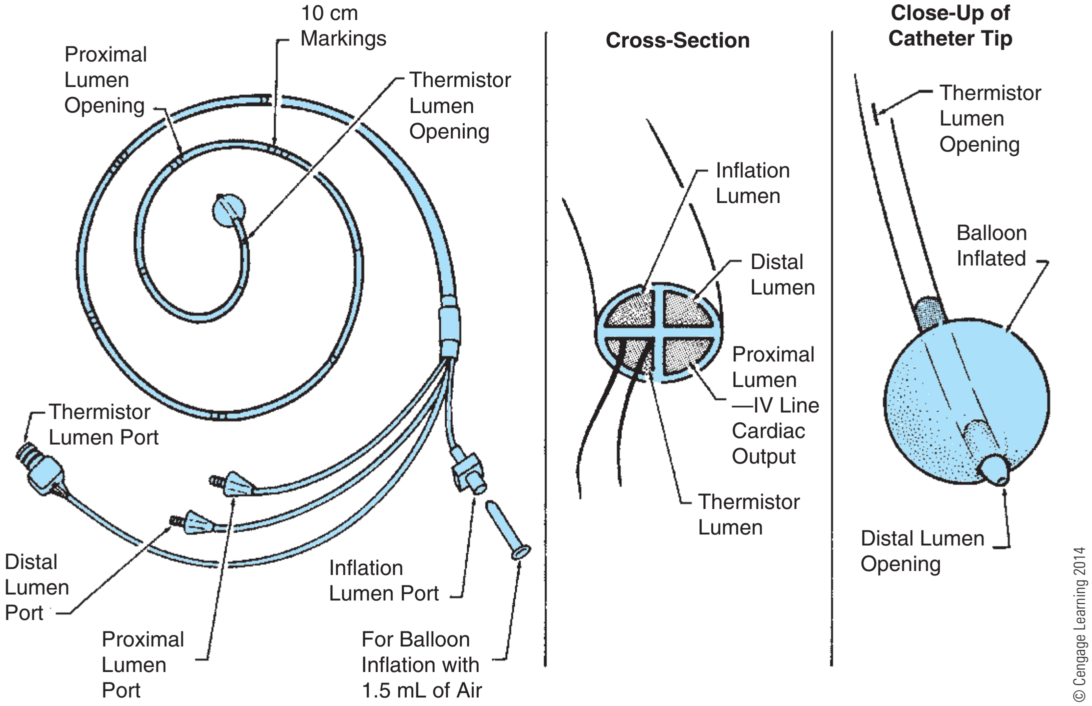
10 cm Markings
Proximal Lumen Opening
Thermistor Lumen Opening
Thermistor Lumen Port
Distal Lumen Port
Proximal Lumen Port
Inflation Lumen Port
For Balloon Inflation with 1.5 mL of Air
Cross-Section
Inflation Lumen
Distal Lumen
Proximal Lumen —IV Line Cardiac Output
Thermistor Lumen
Close-Up of Catheter Tip
Thermistor Lumen Opening
Balloon Inflated
Distal Lumen Opening
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FIGURE 10-6 Components of a Swan-Ganz (pulmonary artery) catheter.

The pulmonary artery catheter is placed within the pulmonary artery, and it can measure the pulmonary arterial pressure (PAP) and the pulmonary capillary wedge pressure (PCWP). Since it is inserted at the same site as the CVP catheter, it has similar complications as well as additional ones related to balloon inflation, such as pulmonary artery hemorrhage and pulmonary infarction.

The pulmonary artery catheter (Figure 10-6) has a number of variations but typically it is 110 cm in length with three lumens (interior channels). The exterior of the catheter is marked off in 10-cm segments by thin and thick black lines to estimate the catheter tip location on insertion. At the tip of the catheter there is an opening (PA distal lumen or port) connected with one lumen. About 30 cm back from the catheter tip there is another opening (proximal injectate port) connected to another lumen. When properly inserted, this proximal port is in the right atrium. Near the catheter tip is a small (1.5 mL maximum inflation volume) balloon connected to a lumen that allows for inflation of the balloon with a syringe. Also at the catheter tip is a thermistor (temperature-sensing device) connected to a wire.

Insertion of Pulmonary Artery Catheter

The pulmonary artery catheter is usually inserted into either the subclavian or internal jugular vein. From there, it is advanced to the superior vena cava and right atrium. The balloon is then inflated and the blood flow moves the catheter with its inflated balloon just as the wind moves a sail. The catheter proceeds to the right ventricle and into the pulmonary artery where it will eventually "wedge" in a smaller branch of the pulmonary artery. The balloon is then deflated and the catheter stabilized in place (Figure 10-7).

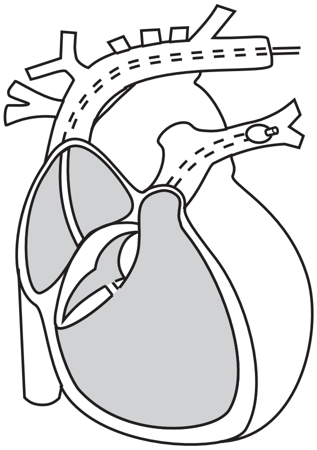

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FIGURE 10-7 Position of a pulmonary artery catheter.

As the pulmonary artery catheter is being inserted, its movement can be followed on the bedside monitor by observing the various pressure waveforms as the catheter passes freely from the right atrium (RA) to a wedged position in the pulmonary artery (Figure 10-8).

The balloon stays deflated and the PAP tracing remains on the monitor at all times. The balloon is inflated only momentarily to measure the pulmonary capillary wedge pressure.

Components of Pulmonary Arterial Pressure Waveform

✓ The systolic component of the PAP waveform may be increased in conditions in which the pulmonary vascular resistance or pulmonary blood flow is increased.

The pulmonary arterial pressure waveform has three components: systolic phase, diastolic phase, and dicrotic notch. The dicrotic notch on the PAP waveform reflects closure of the semilunar valves (mainly the pulmonary valve) at the end of contraction and prior to refilling of the ventricles. The slight elevation seen at the dicrotic notch represents the transient increase in pulmonary artery pressure due to backup of blood flow immediately following closure of the semilunar valves (Figure 10-9).

✓ The dicrotic notch reflects closure of the semilunar valves at the end of contraction and prior to refilling of the ventricles.

Abnormal Pulmonary Artery Waveform. The systolic component of the pulmonary artery pressure waveform may be increased in conditions in which the pulmonary vascular resistance or pulmonary blood flow is increased. Obstruction in the left heart may also cause backup of blood flow in the pulmonary artery and an increase in pulmonary artery pressure (Schriner, 1989). An irregular pressure tracing on the pulmonary artery pressure waveform may be seen in arrhythmias due to changes in diastolic filling time and volume.

PAP Measurements

✓ The normal systolic PAP ranges from 15 to 25 mm Hg and the diastolic PAP from 6 to 12 mm Hg.

Pulmonary arterial pressure (PAP) is measured when the catheter is inside the pulmonary artery with the balloon *deflated*. The normal systolic PAP is about the same as the right ventricular systolic pressure and ranges from 15 to 25 mm Hg. The normal diastolic PAP range is from 6 to 12 mm Hg. Pulmonary hypertension is defined as

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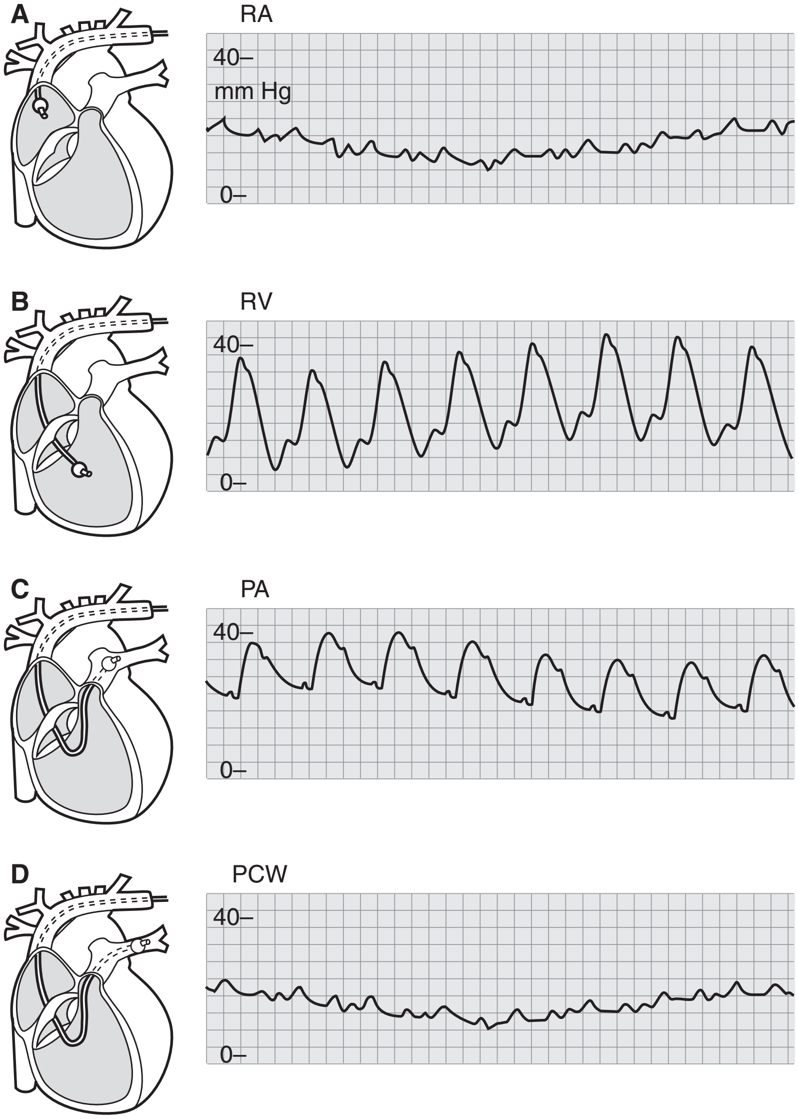

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FIGURE 10-8 Waveform characteristics during advancement of pulmonary artery catheter. (A) Right atrium (RA) and right atrial (central venous) waveform; (B) Right ventricle (RV) and right ventricular waveform; (C) Pulmonary artery (PA) and pulmonary arterial waveform; and (D) Pulmonary capillary wedge (PCW) and pulmonary capillary wedge pressure waveform.

✓ Pulmonary hypertension is defined as a systolic PAP >35 mm Hg, or mean PAP >25 mm Hg at rest (> 30 mm Hg with exertion).

a systolic pulmonary artery pressure of >35 mm Hg or a mean pulmonary artery pressure of >25 mm Hg at rest or >30 mm Hg with exertion (McGoon et al., 2004).

When positive pressure ventilation is augmented with positive end-expiratory pressure (PEEP), the PAP is increased because overdistension of the alveoli compresses the surrounding capillaries and raises the capillary and arterial pressures (Versprille, 1990). Increase in pulmonary vascular resistance or pulmonary blood flow can also lead to an increased PAP, because the pressure measurement is directly related to the resistance and blood flow.

A higher than normal PAP may also be seen in left ventricular dysfunction such as left ventricular failure and mitral valve disease. This is because obstruction or backup

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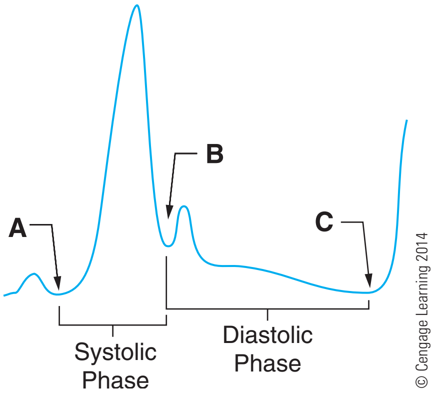

FIGURE 10-9 Pulmonary arterial pressure (PAP) waveform. (A) Beginning systole; (B) Dicrotic notch (closure of aortic valve); and (C) End diastole.

of blood flow in the left heart leads to congestion in the pulmonary circulation. This is reflected as an elevated PAP.

On the other hand, the PAP may be decreased in conditions of hypovolemia or use of mechanical ventilation. When positive pressure ventilation is used on patients who have unstable hemodynamic status, it may lead to a depressed cardiac output, venous return, pulmonary circulating volume, and PAP (Versprille, 1990). The conditions that may affect the PAP are summarized in Table 10-7.

✓ Positive pressure ventilation causes a decrease in the pulmonary arterial pressure.

Effects of Positive Pressure Ventilation. Positive pressure ventilation causes a decrease of the pulmonary arterial pressure (Figure 10-10). This condition is due to decreased venous return to the right ventricle, lower right ventricular output, and lower blood volume (pressure) in the pulmonary arteries (Perkins et al., 1989; Versprille, 1990).

TABLE 10-7 Conditions That Affect the Pulmonary Arterial Pressure

PAP	Conditions	Examples
Increase	Mechanical ventilation*	PEEP
	Increase in pulmonary vascular resistance	Pulmonary embolism Hypoxic vasoconstriction Primary pulmonary hypertension
	Increase in pulmonary blood flow	Hypervolemia Left to right shunt
	Left heart pathology	Left ventricular failure Mitral valve disease
Decrease	Mechanical ventilation*	Positive pressure ventilation
	Decrease in pulmonary blood flow	Hypovolemia

**The effects of mechanical ventilation on the PAP are highly variable, depending on the interaction between the peak inspiratory pressure, PEEP, and the patient's compliance and hemodynamic status.*
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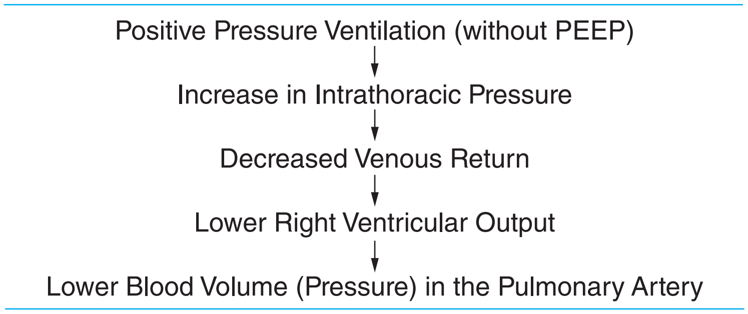
Positive Pressure Ventilation (without PEEP)
Increase in Intrathoracic Pressure
Decreased Venous Return
Lower Right Ventricular Output
Lower Blood Volume (Pressure) in the Pulmonary Artery

Note: When PEEP is used in conjunction with positive pressure ventilation, the PAP may not show a decrease because PEEP tends to increase the PAP by compressing the pulmonary vessels.

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FIGURE 10-10 Effects of positive pressure ventilation.

In the absence of compensation by increasing the heart rate, decrease of right and left ventricular stroke volumes generally leads to a decreased cardiac output.

Pulmonary Capillary Wedge Pressure

✔ PCWP reflects the left ventricular preload.

✔ The PCWP reading is typically taken at end-expiration for both spontaneous breathing and mechanically ventilated patients.

The pulmonary artery catheter is also used to measure the pulmonary capillary wedge pressure (PCWP) (also called pulmonary artery wedge pressure). PCWP is measured by slowly inflating the balloon via the balloon inflation port on the pulmonary artery catheter. As the balloon inflates, the pulmonary arterial waveform on the monitor will change to the wedged pressure waveform. Proper inflation of the balloon usually requires no more than 1.5 mL (0.75 to 1.5 mL depending on size of balloon) of air. The balloon is deflated as soon as the reading of PCWP is obtained.

The PCWP reading is typically taken at end-expiration for both spontaneous breathing and mechanically ventilated patients (Ahrens, 1991; Campbell et al., 1988). This practice should be done consistently for consistent PCWP measurements and meaningful interpretation of hemodynamic data.

Components of Pulmonary Capillary Wedge Pressure Waveform

The components of the PCWP waveform are similar to the CVP or right atrial waveform. When all of the components are present, the *a* wave of the PCWP waveform reflects left atrial contraction and *x* downslope represents the decrease in left atrial pressure following contraction. The *c* wave, if present, is seen along the *x* downslope, and it occurs during closure of the mitral valve. The *v* wave indicates left ventricular contraction and passive atrial filling. The *y* downslope is due to the decrease in blood volume and pressure following the opening of the mitral valve (Figure 10-11).

Abnormal Pulmonary Capillary Wedge Pressure Waveform. Increased PCWP measurements are often observed in conditions where partial obstruction or excessive blood flow is present in the left heart (Schriner, 1989). Two common changes in the PCWP waveform are the *a* and *v* waves.

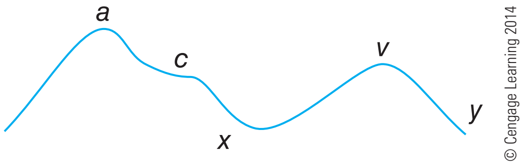

FIGURE 10-11 Pulmonary capillary wedge pressure (PCWP) waveform. a wave: left atrial contraction; c wave (may be absent): closure of mitral valve; x downslope: decreased left atrial pressure following atrial contraction; v wave: left ventricular contraction and passive atrial filling; y downslope: decrease of blood volume (pressure) following the opening of mitral valve.

The *a* wave of the PCWP waveform may be increased in conditions leading to higher resistance to left ventricular filling. Some examples are mitral valve stenosis, left ventricular hypervolemia or failure, and decreased left ventricular compliance.

The *v* wave of the PCWP waveform may be increased due to mitral valve insufficiency. This condition leads to regurgitation (backward flow) of blood from the left ventricle to the left atrium through the incompetent mitral valve.

PCWP Measurements

✓ The normal PCWP ranges from 8 to 12 mm Hg.

✓ In left ventricular failure, the PCWP is usually elevated (≥18 mm Hg) along with a near-normal PAP.

✓ In pulmonary edema where the PCWP is normal, the cause may be acute pulmonary hypertension or an increase in capillary permeability (e.g., ARDS).

The normal range for PCWP is from 8 to 12 mm Hg. Positive pressure ventilation or PEEP can affect wedge pressure readings since over distension of the alveoli compresses the surrounding capillaries and raises the capillary and arterial pressures. A higher than normal wedge pressure may also be seen in left ventricular dysfunction. This is because left ventricular failure causes backup of blood flow in the left heart and pulmonary circulation. A PCWP reading of ≥18 mm Hg along with a near-normal PAP suggests presence of left ventricular dysfunction or left heart failure.

The PCWP measurement may be used to distinguish cardiogenic and noncardiogenic pulmonary edema. In pulmonary edema that is caused by left ventricular failure, the PCWP is usually elevated (≥18 mm Hg) along with a near-normal PAP. In pulmonary edema where the PCWP is normal, the cause may be acute pulmonary hypertension or an increase in capillary permeability (e.g., ARDS). The conditions that may affect the PCWP measurements are outlined in Table 10-8.

TABLE 10-8 Conditions That Affect the Pulmonary Capillary Wedge Pressure

PCWP	Conditions	Examples
Increase	Increase in pulmonary blood flow	Hypervolemia
	Left heart pathology	Left ventricular failure; Mitral valve disease
	Mechanical factor	Overwedging of balloon
Decrease	Mechanical ventilation	PEEP
	Decrease in pulmonary blood flow	Hypovolemia

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Verification of the Wedged Position

✓ The wedged position of a pulmonary artery catheter may be confirmed by: (1) PAP diastolic-PCWP gradient; (2) postcapillary-mixed venous PO_2 gradient; and (3) postcapillary-mixed venous O_2 saturation gradient.

Since artifact or dampened waveform may occur during inflation of the balloon, and it resembles that of a wedged pressure tracing, using the PCWP tracing alone on the monitor to verify the wedged position may not be always reliable. Three methods are available to confirm a properly wedged pulmonary artery catheter: (1) PAP diastolic-PCWP gradient; (2) postcapillary-mixed venous PO_2 gradient; and (3) postcapillary-mixed venous O_2 saturation gradient.

PAP Diastolic-PCWP Gradient. Under normal conditions, the PAP diastolic value is about 1 to 4 mm Hg higher than the average wedge pressure of the same individual (Daily et al., 1985). However, the PAP diastolic value may be lower than actual with forceful spontaneous inspiratory efforts. The PCWP may be higher than actual if there is significant downstream obstruction such as mitral valve disease (McGrath, 1986). These factors must be taken into account when evaluating the pressure gradient between PAP diastolic pressure and PCWP.

Postcapillary-Mixed Venous PO_2 Gradient. The PO_2 of a blood gas sample from the distal opening of a properly wedged catheter should be at least 19 mm Hg higher than that from a systemic artery. The PCO_2 should be at least 11 mm Hg lower. These differences are expected because a properly wedged catheter does not allow mixing of shunted venous blood with the postcapillary (oxygenated) blood. This procedure may not be feasible for a hypovolemic patient because up to 40 mL of waste (mixed venous) blood sample may be required before reaching the postcapillary blood sample (Morris et al., 1985).

Postcapillary-Mixed Venous O_2 Saturation Gradient. If the pulmonary artery catheter is capable of monitoring oxygen saturation by the oximetry method, the oxygen saturation value of a properly wedged catheter should be about 20% higher than the one recorded with the balloon deflated (Morris et al., 1985).

Cardiac Output and Cardiac Index

Another important value of the pulmonary artery catheter is its ability to measure cardiac output by the thermodilution method. During cardiac output measurement, a small amount (10 mL) of iced or room-temperature fluid (usually 5% dextrose in water, D5W) is injected into the proximal port of the pulmonary artery catheter. The temperature change of the blood flow is recorded as the flow passes by the thermistor at the catheter tip. This and other measurements are computed and the flow rate through the heart is displayed as cardiac output. The normal cardiac output for an adult is from 4–8 L/min.

✓ The normal cardiac output for an adult is from 4 to 8 L/min.

Current pulmonary artery catheters are capable of monitoring cardiac output continuously by thermodilution without injecting a bolus of room temperature or iced injectate. This technology uses a thermal strip on the outside of the catheter which is slightly heated by an electronic signal.

✓ The normal cardiac index is 2.5 to 3.5 $L/min/m^2$.

Since cardiac output normally varies from person to person depending on the size of the individual, it is common to "index" the value by dividing cardiac output

TABLE 10-9 Ventricular Preloads and Afterloads

Main Device (Measurement)	Implication	Examples
Arterial catheter (Left ventricular afterload)	Condition of systemic arterial pressure	Arterial pressure is increased in systemic hypertension or fluid overload. Arterial pressure is decreased in systemic hypotension or fluid depletion.
Central venous catheter (Right ventricular preload)	Condition of systemic venous return	Central venous pressure (CVP) is increased in systemic hypertension or hypervolemia. CVP is decreased in systemic hypotension or hypovolemia.
Pulmonary artery catheter (Right ventricular afterload)	Condition of pulmonary artery	Pulmonary artery pressure (PAP) is increased in pulmonary hypertension or blood flow obstruction in left heart (e.g., mitral valve stenosis). PAP is decreased in pulmonary hypoperfusion as in right-sided heart failure.
Pulmonary artery catheter (Balloon inflated) (Left ventricular preload)	Condition of left heart	Pulmonary capillary wedge pressure (PCWP) is increased in left heart flow obstruction. PCWP is decreased in severe hypotension or dehydration.

cardiac index (C.I): A cardiac output measurement relative to a person's body size.

(C.O.) by body surface area (BSA). The **cardiac index** (C.I.) is normally 2.5 to 3.5 L/min/m^2 and it is calculated as follows:

$$C.I. = C.O. / BSA$$

SUMMARY OF PRELOADS AND AFTERLOADS

Each ventricle has its own preload and afterload measurements. Their meaning and common pathologic implications are summarized in Table 10-9.

CALCULATED HEMODYNAMIC VALUES

systemic vascular resistance: Resistance of the arterial system into which the left heart is pumping. Normal range is 800–1,500 dynes.sec/cm^5.

From the CVP, PAP, and other related measurements, the following parameters may be calculated: stroke volume and stroke volume index, oxygen consumption and oxygen consumption index, pulmonary vascular resistance, and **systemic vascular resistance.**

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Stroke Volume and Stroke Volume Index

Stroke volume (S.V.) is calculated by dividing the cardiac output (C.O.) by the heart rate (HR). The stroke volume index is calculated by dividing the stroke volume by the body surface area (BSA).

$$\text{S.V.} = \frac{\text{C.O.}}{\text{HR}}$$

$$\text{S.V.I.} = \frac{\text{S.V.}}{\text{BSA}}$$

contractility: Pumping strength of the heart. Contractility may be increased by improving the blood volume or by positive inotropic medications.

✓ The stroke volume is determined by three factors: contractility, preload, and afterload.

The stroke volume is determined by three factors: **contractility,** preload, and afterload. Contractility is the pumping strength of the heart. Some conditions that may lower the contractility of the heart include extremes of myocardial compliance (too high or too low), and excessive end-diastolic volume. Preload is the end-diastolic stretch of cardiac muscle fiber, expressed in pressure units (mm Hg or cm H_2O). Hypovolemia and shock are two conditions that usually cause a decreased preload. Afterload is the tension or pressure that develops in the ventricle during systole (contraction), expressed in pressure units (mm Hg or cm H_2O). Afterload is usually increased in conditions of downstream flow obstruction or excessive volume.

Oxygen Consumption and Oxygen Consumption Index

The oxygen consumption reflects the amount of oxygen consumed in one min. The oxygen consumption index reflects the amount of oxygen used relative to the body size. They are calculated as follows:

$$\dot{V}O_2 = Q_T \times C(a\text{-}\dot{v})O_2$$

$$\dot{V}O_2 \text{ index} = \frac{\dot{V}O_2}{\text{BSA}}$$

Pulmonary Vascular Resistance

pulmonary vascular resistance (PVR): Resistance of the arterial system into which the right heart is pumping. Normal range is 50–150 dynes.sec/cm^5.

The **pulmonary vascular resistance (PVR)** measures the blood vessel resistance to blood flow in the pulmonary circulation. For example, PVR is elevated in pulmonary hypertension or left heart obstruction (e.g., mitral valve stenosis).

$$\text{PVR} = \frac{(\text{PAP} - \text{PCWP}) \times 80}{\text{C.O.}}$$

Systemic Vascular Resistance

The systemic vascular resistance (SVR) measures the blood vessel resistance to blood flow in the systemic circulation. For example, SVR is elevated in systemic hypertension.

$$\text{SVR} = \frac{(\text{MAP} - \text{RAP}) \times 80}{\text{C.O.}}$$

MIXED VENOUS OXYGEN SATURATION

A special version of the pulmonary artery catheter uses fiberoptic technology to monitor the mixed venous oxygen saturation ($S\dot{v}O_2$). The fiberoptic central venous catheter measures the $S\dot{v}O_2$ accurately within the clinical range (between 50% and 80%) (Fletcher, 1988). When $S\dot{v}O_2$ is used with other monitoring capabilities of the pulmonary artery catheter, it can provide valuable information concerning oxygen delivery and consumption.

Decrease in Mixed Venous Oxygen Saturation

✓ The normal $S\dot{v}O_2$ is about 75%. $S\dot{v}O_2$ measurements from 50% to 70% indicate decreasing oxygen delivery (↓DO_2) or increasing oxygen consumption (↑VO_2) with compensatory O_2 extraction.

For individuals with a balanced oxygen delivery (DO_2) and oxygen consumption ($\dot{V}O_2$), the measured $S\dot{v}O_2$ is between 68% and 77% with an average of 75%. $S\dot{v}O_2$ measurements from 50% to 70% indicate decreasing DO_2 or increasing $\dot{V}O_2$ with compensatory O_2 extraction—a process to meet the minimal oxygen needs by the body. When the $S\dot{v}O_2$ drops to a range of 30%–50%, lactic acidosis becomes evident due to exhausting of extraction. From 25% to 30%, severe lactic acidosis is common. Below 25%, cellular death is ensured (Zaja, 2007).

Common causes of decreased $S\dot{v}O_2$ due to poor oxygen delivery include low cardiac output, anemia, and hypoxic hypoxia. Causes of decreased $S\dot{v}O_2$ due to excessive oxygen consumption include fever, seizures, increased physical activity or work of breathing, stress, and pain. Some conditions that may lead to a decreased $S\dot{v}O_2$ are summarized in Table 10-10.

Increase in Mixed Venous Oxygen Saturation

✓ Increases in $S\dot{v}O_2$ above 75% are uncommon but may occur when the tip of the pulmonary artery catheter is improperly wedged.

Increases in $S\dot{v}O_2$ above 75% are uncommon but may occur when the tip of the pulmonary artery catheter is improperly wedged. Once in this abnormal position, the forward mixed venous blood flow is obstructed while the catheter tip senses the blood from an area with a high ventilation/perfusion ratio, and therefore a high oxygen saturation. Other conditions that reduce metabolic oxygen consumption may also lead to an increase in $S\dot{v}O_2$. Some examples include use of analgesics or sedatives, full ventilatory support on mechanical ventilation, and hypothermia (Zaja, 2007).

In some uncommon conditions, an increased $S\dot{v}O_2$ may occur to patients with sepsis or cyanide poisoning in which the tissues cannot fully utilize oxygen. The mechanism of hypoxia for sepsis is due to peripheral shunting. Cyanide poisoning causes histotoxic hypoxia that renders the tissues unable to carry out normal aerobic metabolism. These patients may have normal PaO_2, SaO_2, CaO_2, and oxygen transport, but they are often hypoxic. A plasma lactate concentration of greater than 10 mEq/L in smoke inhalation or greater than 6 mEq/L after reported or strongly suspected pure cyanide poisoning suggests significant cyanide exposure (Leybell et al., 2011). Some conditions that may lead to an increased $S\dot{v}O_2$ are summarized in Table 10-10.

TABLE 10-10 Conditions That Affect the $S\dot{v}O_2$ Measurement

$S\dot{v}O_2$	Conditions	Examples
Decrease	Poor oxygen delivery	Low cardiac output Anemia Hypoxic hypoxia
	Excessive oxygen consumption	Fever Seizures Increased metabolic rate Increased physical activity Stress Pain
	Depletion of venous oxygen reserve	Severe and prolonged hypoxia
Increase	Technical problem	Improperly wedged catheter
	Increase in oxygen delivery	↑ Cardiac output ↑ CaO_2
	Impaired oxygen utilization	Sepsis Cyanide poisoning
	Decrease in oxygen consumption	Hypothermia Postanesthesia Pharmacologic paralysis

LESS-INVASIVE HEMODYNAMIC MONITORING

A number of less-invasive techniques for obtaining hemodynamic data have been developed over the past decade. Pulse contour analysis is considered a less-invasive technique because it requires an indwelling arterial catheter.

Pulse Contour Analysis

Earlier in this chapter, the shape and significance of the arterial pressure wave form was discussed. The difference between peak systolic pressure and end-diastolic pressure on this waveform is known as pulse pressure. **Pulse contour analysis** (also known as arterial pressure waveform analysis) uses an arterial catheter and other data to derive the cardiac output. This is done by special algorithms using the arterial pressure waveform, arterial vascular compliance, and specific patient data to calculate the stroke volume and stroke volume index. The stroke volume and stroke volume index are multiplied by the heart rate to yield the cardiac output and cardiac index.

pulse contour analysis: A less-invasive method to calculate the stroke volume and stroke volume index by using the area under the arterial pressure waveform and specific patient data.

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✓ **Pulse contour analysis** uses the arterial pressure waveform, arterial vascular resistance and patient data to calculate the stroke volume and cardiac output.

Pulse contour analysis is not entirely noninvasive because an arterial catheter is required, and some systems also require a central venous catheter. There are a number of monitoring systems based on pulse contour analysis. Since the arterial pressure waveform varies with changes in arterial compliance, patient condition, and medications, the systems must be calibrated with another reference standard. Two common reference standards are lithium dilution (Pittman et al., 2005) and transpulmonary thermodilution (Della et al., 2002).

The Lithium Dilution Cardiac Output (LiDCO) system uses a peripheral venous catheter into which lithium chloride is injected and then the lithium concentration is measured at the arterial catheter. The Pulse Contour Cardiac Output (PiCCO) system uses a combination of the transpulmonary thermodilution technique and arterial pulse contour analysis. Transpulmonary thermodilution is done by injecting a cold saline solution into a central venous line and then the temperature is measured at the arterial side (typically via a femoral artery line).

In both LiDCO and PiCCO systems the cardiac output measurement needs to be repeated on a regular basis and in the occurrence of any changes in patient condition or fluid and vasoactive drug administration.

The FLOTRAC system does not require calibration with some other method of measuring cardiac output, but it uses a transducer attached to the patient's peripheral arterial line and interfaced with a special monitor (Vigileo) to measure pulse pressure. It also uses customized patient data and algorithms to account for changes in arterial compliance and resistance. The data are updated every 20 seconds and displayed as a continuous value. This system does not require a central venous line but there is a specially adapted fiberoptic CVP (PreSep) line which can interface with the same Vigileo monitor to provide central venous oxygen saturation data ($S\dot{v}O_2$) to complement the continuous cardiac output data.

NONINVASIVE HEMODYNAMIC MONITORING

There are three major types of noninvasive hemodynamic monitoring methods: transesophageal echocardiography, carbon dioxide elimination ($\dot{V}CO_2$), and impedance cardiography (ICG). Following is a discussion of each technology.

Transesophageal Echocardiography

transesophageal echocardiography: A method using a Doppler transducer in the esophagus for an indirect measurement of the blood flow velocity in the descending aorta and the calculation of the cardiac output and other hemodynamic data.

Transesophageal echocardiography provides diagnosis and monitoring of many structural and functional abnormalities of the heart. It can also be used to calculate cardiac output from measurement of blood flow velocity by recording the Doppler shift of ultrasound. The time velocity integral obtained for the blood flow in the left ventricular outflow tract (e.g., descending aorta) is multiplied by the cross-sectional area and the heart rate to yield the cardiac output. This Doppler technique requires a highly skilled technician to obtain accurate readings (Mark et al., 1986). The transesophageal echocardiography procedure may be done at the bedside, and continuous readings are available with this procedure. A Doppler transducer probe

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is placed into the esophagus (via the mouth or nose) with its distal end resting at the midthoracic level. The probe is rotated until it faces the aorta and is able to pick up the aortic blood flow signal. In three studies, the cardiac output measured by this technique correlates well with the measurements using the traditional thermodilution method (DiCorte et al., 2000; Perrino et al., 1998; Mark et al., 1986).

Carbon Dioxide Elimination ($\dot{V}CO_2$)

carbon dioxide elimination ($\dot{V}CO_2$): A technology to monitor and measure cardiac output based on changes in respiratory CO_2 concentration during a period of rebreathing.

Carbon dioxide elimination ($\dot{V}CO_2$) is a technology that can monitor and measure cardiac output based on changes in respiratory CO_2 concentration during a brief period of rebreathing. The NICO$_2$® (with cardiac output option) is a cardiopulmonary management system that incorporates different sensors to measure the flow, airway pressure, and CO_2 concentration. These measurements are used to calculate CO_2 elimination. A Fick partial rebreathing method is used to derive the cardiac output.

The original Fick method uses the oxygen consumption ($\dot{V}O_2$) and arterial-mixed venous oxygen content difference ($C_{(a-v)}O_2$) to calculate the cardiac output. (C.O. = $\dot{V}O_2$ / $C_{(a-v)}O_2$). This method for calculating cardiac output requires the use of specialized equipment and has never been suitable in the traditional clinical setting. The NICO$_2$® uses $\dot{V}CO_2$ instead of $\dot{V}O_2$. End-tidal CO_2 from an exhaled breath sample is used instead of using mixed venous and arterial blood samples (for $C_{(a-v)}O_2$). The NICO® system (Respironics®) can provide continuous cardiac output noninvasively via this method.

IMPEDANCE CARDIOGRAPHY

impedance cardiography (ICG): A noninvasive procedure to measure or trend the hemodynamic status of a patient.

Impedance cardiography (ICG), also called thoracic electrical bioimpedance (TEB), is a major division of noninvasive technique for hemodynamic monitoring. ICG is based on a technology originally used by NASA in the 1960s. The introduction of the microprocessor and the working knowledge of echocardiography and magnetic resonance imaging make ICG possible. ICG is a noninvasive procedure to measure or trend the hemodynamic status of a patient in clinical settings ranging from critical care to outpatient care. Several noninvasive ICG devices are available and each offers different technology to measure and calculate the hemodynamic values.

The *IQ* system (Wantagh Incorporated, Bristol, MA) uses a patented signal processing technique to identify the opening and closing of the aortic valve for the precise measurement of the ventricular ejection time (VET). Another device incorporates "ensemble averaging" to estimate the VET by using the QRS of the ECG and the raw dZ/dt (change in impedance/time) waveform (SORBA Medical Systems, Inc., Brookfield, WI). A third manufacturer of ICG (BioZ System, CardioDynamics, San Diego, CA) uses digital signal processing and an R-wave detection system to establish the dZ/dt. ICG has proven to be a simple and accurate method to measure and monitor a patient's hemodynamic status (Clancy et al., 1991).

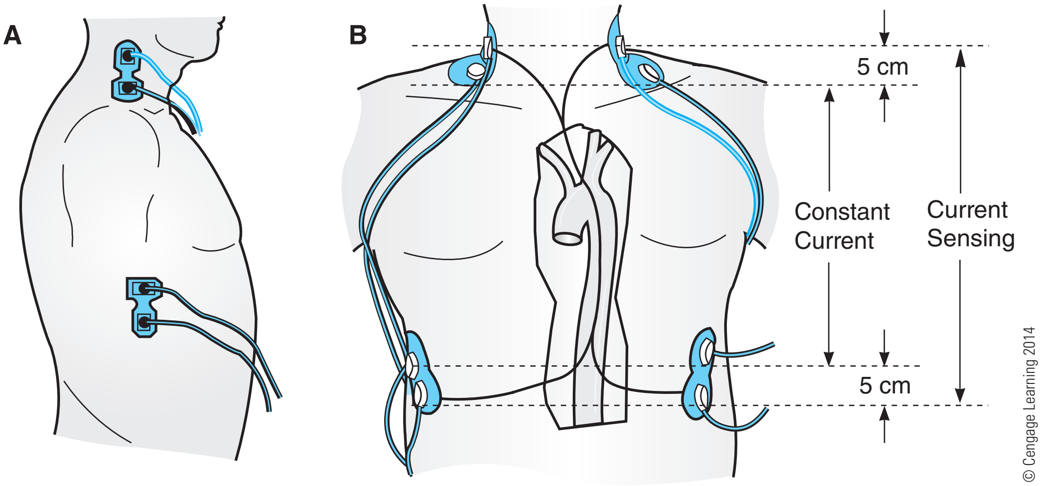

FIGURE 10-12 Typical placement of impedance cardiography (ICG) electrodes

Theory of Operation

ICG uses external electrodes to input a high frequency, low amplitude current and measure changes of electrical resistance (impedance) in the thorax.

ICG uses external electrodes to input a high frequency, low amplitude current and to measure changes of electrical resistance (impedance) in the thorax. In a typical setup, four outer and four inner electrodes are placed on the patient, as shown in Figure 10-12. The outer electrodes transmit a constant, low amplitude electrical current through the thorax. The inner electrodes measure the impedance (resistance) to the electrical signal according to the changing blood flow in the aorta.

Since the impedance changes reflect the blood flow in the ascending aorta during systole and asystole, the changes in blood velocity are calculated and reported as values for different hemodynamic parameters.

The volume and velocity of blood flow in the ascending aorta changes with each cardiac cycle—increasing volume and velocity during systole and decreasing volume and velocity during asystole. Since the impedance changes reflect the blood flow in the ascending aorta, the changes in blood velocity are calculated and reported as values for different hemodynamic parameters. Figure 10-13 shows an example of the impedance cardiography waveforms.

Thermodilution Method and ICG

Thermodilution is the most commonly used invasive technique for measuring and calculating the hemodynamic values. The accuracy and reliability of this method rely on the proper (and correct) computation constant, injectate volume, injectate temperature measurement, injection technique, timing of injection, and averaging strategies (Wantagh Inc., 2004). Since the thermodilution method provides hemodynamic measurements in a limited time frame, it cannot be used to monitor the dynamic nature of the cardiovascular system.

The noninvasive nature of ICG makes it an ideal tool to monitor a patient's hemodynamic status. Some of the measured and calculated hemodynamic parameters provided by ICG include: cardiac output, cardiac index, stroke volume, stroke volume

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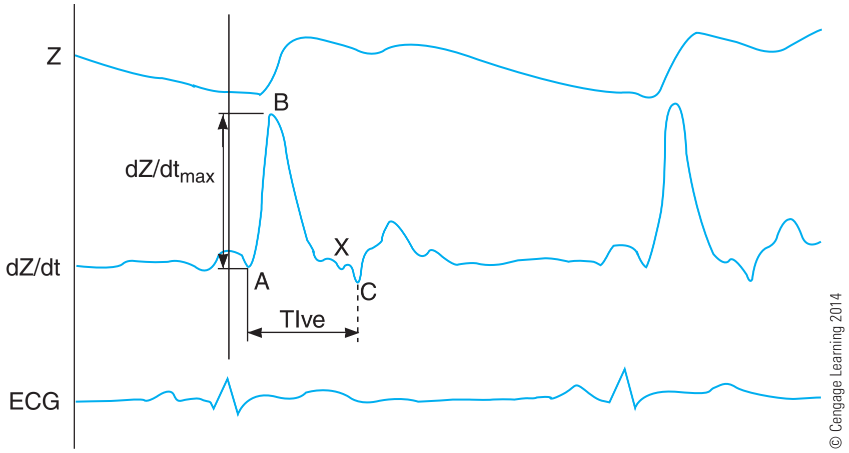

FIGURE 10-13 Impedance cardiography waveforms. Z—pulse contour curve; dZ/dt—impedance curve; ECG—electrocardiogram curve; dZ/dt$_{max}$—maximum value of the first derivative of the impedance curve; A—opening of the aortic valve; B—maximum systolic value; X—closing of the aortic valve; C—opening of the pulmonic valve; Tlve—left ventricle ejection time.

✓ Some parameters provided by ICG include: cardiac output, cardiac index, stroke volume, stroke volume index, systemic vascular resistance, systemic vascular resistance index, contractility, and fluid status.

index, systemic vascular resistance, systemic vascular resistance index, contractility, and fluid status. Table 10-11 lists some hemodynamic parameters provided by ICG.

Unlike the thermodilution method in which a pulmonary artery catheter is required, ICG cannot provide the values for pulmonary artery pressure, pulmonary artery wedge pressure, pulmonary vascular resistance, and pulmonary vascular resistance index.

TABLE 10-11 Hemodynamic Parameters Provided by Impedance Cardiography

Measured	Calculated
Heart rate	Stroke volume/index
Thoracic fluid content	Cardiac output/index
Mean arterial pressure[a]	Systemic vascular resistance/index
Acceleration index[b]	Left cardiac work/index
Velocity index[c]	Left stroke work/index
Pre-ejection period[d]	
Left ventricular ejection time[e]	

[a] *If automatic blood pressure (oscillometric method) is used.*

[b] *Acceleration of blood flow in the aorta within the first 10 to 20 m/sec after the opening of the aortic valve.*

[c] *Peak blood flow velocity in the aorta.*

[d] *Time interval from the beginning of electrical stimulation of the ventricles to the opening of the aortic valve (electrical systole).*

[e] *Time interval from the opening to the closing of the aortic valve (mechanical systole).*

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Accuracy of ICG

✔ The correlation of cardiac output determined by ICG versus the Fick method and the thermodilution method were 0.84 and 0.80, respectively.

Many studies have been done to compare and validate the accuracy of ICG with other methods of hemodynamic monitoring (Drazner et al., 2002; Ziegler et al., 1999). In a study of patients with pulmonary arterial hypertension, the correlation of cardiac output determined by ICG versus the Fick method and the thermodilution method were 0.84 and 0.80, respectively (Yung et al., 2004). These correlation indices are similar to the results of other studies using different patient populations. Since ICG is less variable and more reproducible than other invasive methods, it has shown sufficient clinical usefulness to become a standard practice in noninvasive hemodynamic evaluations (Van De Water et al., 2003).

✔ Technical and measurement errors of ICG include: wrong placement of electrodes, abnormal body structure, tachycardia, presence of pacemaker, arrhythmias, open-heart or aorta surgery, abnormal cardiac anatomy, abnormal hematocrit, and pleural effusion.

Methodology Errors. While ICG is useful in many clinical situations, there are some technical reasons and conditions that may influence the use and accuracy of ICG (Braždžionytė et al., 2004a). They include wrong placement of electrodes; abnormal body structure (cachetic or obese); tachycardia (>120/min); presence of pacemaker; arrhythmias; open-heart or aorta surgery; abnormal cardiac anatomy (e.g., transpositions, aneurysms); abnormal hematocrit; and pleural effusion.

Clinical Application

✔ ICG provides these advantages: noninvasive continuous monitoring, rapid diagnosis and assessment of cardiopulmonary status, hemodynamic response to fluids and drugs, and availability outside the critical care area.

With ICG, the therapeutic effects of fluid administration and resuscitation can be assessed by monitoring the stroke volume and cardiac output. ICG has also been used to evaluate the hemodynamic status of critically ill patients in the intensive care units, surgical areas, and outpatient and emergency departments (Bishop et al., 1996; Milzman et al., 1997; Shoemaker et al., 1994; Wo et al., 1995; Yancey, 2003). Evaluation and follow-up of patients with acute myocardial infarction is also possible with ICG (Braždžionytė et al., 2004b).

In subacute care, adjustment of the dosages of cardiovascular drugs can be done by monitoring the thoracic fluid status, stroke volume, and cardiac output (Franz, 1996). Since ICG monitoring is noninvasive, it can be used in outpatients as well as patients at home. Some advantages of ICG are listed in Table 10-12.

TABLE 10-12 Advantages of Impedance Cardiography

Reduces risk associated with invasive hemodynamic monitoring procedures
Provides rapid diagnosis and assessment of cardiopulmonary status
Offers continuous noninvasive hemodynamic monitoring
Monitors patient's hemodynamic response to fluids and drugs
Reduces use and risk associated with PA catheterization
Reduces cost over invasive hemodynamic monitoring procedures
Provides availability outside the hospital

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SUMMARY

In order to monitor and use hemodynamic waveforms efficiently, one must remember the normal values and recognize the characteristics of normal waveforms. It is only with constant clinical practice that one may learn the intricacies and interactions of those variables that affect hemodynamic waveforms.

It is also essential to realize that monitoring of hemodynamic waveforms should be done on a continuous basis. Impedance cardiography makes this possible. Isolated and independent observations are of limited value, because they do not provide a trend of changing events or patient conditions.

Self-Assessment Questions

1. In hemodynamic monitoring, the pressure measurement made inside a blood vessel is *least* dependent on:
   - A. blood volume.
   - B. blood flow.
   - C. size of blood vessel.
   - D. barometric pressure.

2. During hemodynamic monitoring, the transducer, catheter, and measurement site are usually aligned at the same level. This is done to ensure that the measurements are not affected by the effect of:
   - A. barometric pressure.
   - B. gravity.
   - C. fluid level.
   - D. transducer sensitivity.

3. In the arterial pressure waveform shown, the dicrotic notch is labeled as ___ and it is caused by the ___. (See Figure 10-14.)
   - A. A, opening of the aortic valve
   - B. B, closure of the mitral valve
   - C. B, closure of the aortic valve
   - D. C, opening of the mitral valve

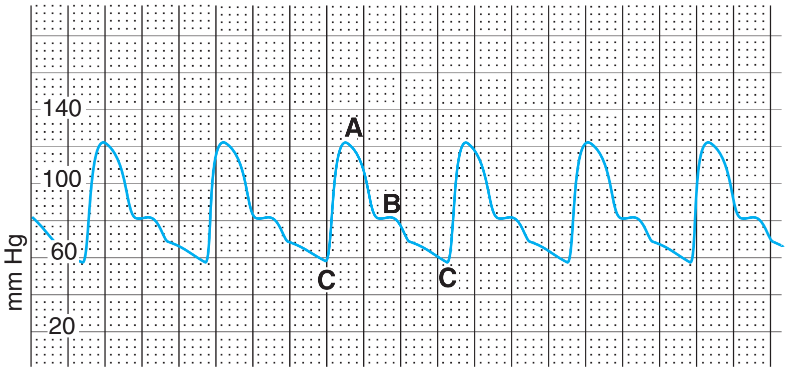

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FIGURE 10-14 Arterial pressure waveform.

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4. The arterial pressure waveform shows a decreasing systolic pressure and a stable diastolic pressure. Based on these two pressure measurements, the pulse pressure is ________ . This condition may be caused by ________ .

   A. decreasing, shock
   B. increasing, hypervolemia
   C. increasing, bradycardia
   D. decreasing, arteriosclerosis

5 to 7. Matching: Refer to the central venous pressure waveform (Figure 10-15) and match the wave or slope with the proper events during a cardiac cycle. Use only three of the five answers provided.

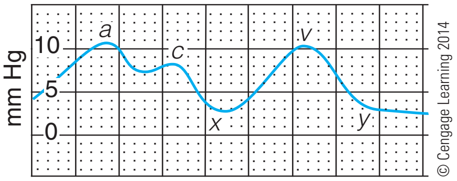

FIGURE 10-15 Central venous pressure waveform.

Wave or Slope	Corresponding Event
5. *a*	A. Ventricular relaxation
6. *c*	B. Atrial contraction
7. *v*	C. Ventricular contraction
	D. Relaxation of right atrium
	E. Closure of tricuspid valve during systole

8. The central venous pressure readings of a patient have been decreasing from an average of 6 to 2 mm Hg. This condition may be caused by all of the following conditions *except*:

   A. positive pressure ventilation.
   B. blood or fluid depletion.
   C. shock.
   D. vasodilation.

9. Which of the following outlines the correct sequence for the placement of a Swan-Ganz catheter?

   A. femoral vein, left atrium, left ventricle, pulmonary artery
   B. internal jugular vein, left atrium, left ventricle, pulmonary artery
   C. femoral artery, right atrium, right ventricle, pulmonary artery
   D. subclavian vein, right atrium, right ventricle, pulmonary artery

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10 to 13. Matching: Match the type of catheter with the intended hemodynamic measurements.

Catheter	Hemodynamic Measurement
10. Arterial catheter	A. Left ventricular preload
11. Central venous catheter	B. Left ventricular afterload
12. Pulmonary artery catheter with balloon deflated	C. Right ventricular preload
13. Pulmonary artery catheter with balloon inflated	D. Right ventricular afterload

14. Mr. Jones, a patient in the coronary care unit being treated for congestive heart failure, has a pulmonary artery catheter in place and the $S\dot{v}O_2$ is 55%. This $S\dot{v}O_2$ value is ___ and it may be caused by ___.

    A. too high, increase in cardiac output
    B. too high, increase in peripheral oxygen consumption
    C. too low, decrease in cardiac output
    D. too low, decrease in peripheral oxygen consumption

15. ________ is a noninvasive monitoring technique that measures the blood flow velocity in the descending aorta to calculate the stroke volume and cardiac output.

    A. Impedance cardiography
    B. Esophageal Doppler ultrasound
    C. Pulse contour analysis
    D. Carbon dioxide elimination

16. Impedance cardiography (ICG) is a noninvasive technique capable of monitoring all of the following hemodynamic values *except:*

    A. thoracic fluid volume.
    B. cardiac output.
    C. pulmonary artery pressure.
    D. systemic vascular resistance.

Answers to Self-Assessment Questions

1. D.
2. B.
3. C.
4. A.
5. B.
6. E.
7. C.
8. A.
9. D.
10. B
11. C.
12. D.
13. A.
14. C.
15. B.
16. C.

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Chapter 11

Ventilator Waveform Analysis

Frank Dennison

Outline

- Introduction
- Flow Waveforms during Positive Pressure Ventilation
- Effects of Constant Flow during Volume-Controlled Ventilation
  - *Flow-Time Waveform*
  - *Pressure-Time Waveform*
  - *Controlled Mandatory Ventilation*
  - *Assist Mandatory Volume-Controlled Ventilation*
  - *Mathematical Analysis of Constant-Flow Ventilation*
- Spontaneous Ventilation during Mechanical Ventilation
  - *Synchronized Intermittent Mandatory Ventilation*
  - *Continuous Positive Airway Pressure*
- Effects of Flow, Circuit, and Lung Characteristics on Pressure-Time Waveforms
  - *Flow and Transairway Pressure*
  - *Compliance and Alveolar Pressure*
- Effects of Descending Ramp Flow Waveform during Volume-Controlled Ventilation
  - *Time- and Flow-Limited Ventilation*
  - *Peak Flow and Tidal Volume Relationship in Time-Limited Ventilation*
  - *Effects of End-Flow on End-Transairway Pressure*
  - *Distribution of Delivered Tidal Volume*
  - *CMV during Descending Ramp Flow Ventilation*
- Waveforms Developed during Pressure-Controlled Ventilation
  - *Pressure-Controlled Ventilation (PCV)*
  - *Assist Breaths during Pressure-Controlled Ventilation*
  - *Inverse Ratio Pressure-Controlled Ventilation (IRPCV)*
- Pressure Support and Spontaneous Ventilation
  - *Pressure Support Ventilation (PSV)*
  - *Adjusting Rise Time during PSV*
  - *SIMV (CFW) and PSV*
  - *SIMV (DRFW) and PSV*
- Effects of Lung Characteristics on Pressure-Controlled Ventilation Waveforms
- Using Waveforms for Patient-Ventilator System Assessment

Patient-Ventilator Dyssynchrony
Dyssynchrony during Constant Flow Ventilation
Dyssynchrony during Descending Ramp Flow Ventilation
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Lack of Ventilator Response
Circuit Leaks
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Pressure-Volume Loop (PVL)
Effects of Lung-Thorax Compliance on PVL
Effects of Airflow Resistance on PVL
Lower Inflection Point on PVL and Titration of PEEP
Upper Inflection Point on PVL and Adjustment of V_T
Effects of Airway Status on Flow-Volume Loop (FVL)
Summary
Self-Assessment Questions
Answers to Self-Assessment Questions
References
Additional Resources

Key Terms

airway opening pressure (P_{AO})
alveolar pressure (P_{ALV})
constant flow waveform (CFW)
controlled mandatory ventilation (CMV)
descending ramp flow waveform (DRFW)
expiratory time (T_E)
flow-volume loop (FVL)
inspiratory time (T_I)
inverse ratio pressure-controlled ventilation (IRPCV)
lung-thorax compliance (C_{LT})
peak alveolar pressure (Peak P_{ALV}; plateau pressure)
peak inspiratory pressure (PIP)
plateau pressure (P_{PLAT}; peak alveolar pressure)
pressure-controlled ventilation (PCV)
pressure-volume loop (PVL)
synchronized intermittent mandatory ventilation (SIMV)
tidal volume (V_T)
total cycle time (TCT)
transairway pressure (P_{TA})
volume-controlled ventilation (VCV)

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Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Describe the waveform characteristics of constant flow during volume-controlled ventilation to include flow-time, pressure-time, and controlled mandatory ventilation.
- Describe the waveform characteristics of spontaneous breathing during mechanical ventilation.
- Provide examples to show the effects of flow, circuit, and lung characteristics on the pressure-time waveform.
- Describe the effects of descending ramp flow during volume-controlled ventilation.
- Describe the waveform characteristics of pressure-controlled ventilation (PCV) and contrast PCV with volume-controlled ventilation.
- Describe the waveform characteristics of pressure-supported ventilation.
- Explain the effects of changing lung characteristics on the PCV waveforms.
- Analyze pertinent waveforms to identify and correct the following: patient-ventilator dyssynchrony, increased airway resistance, loss of elastic recoil, decreased lung-thorax compliance, gas trapping, lack of ventilator response, and circuit leaks.
- Analyze the pressure-volume loop and flow-volume loop to evaluate the changes in compliance and airway resistance.
- Identify the upper and lower inflection points and describe the respective clinical application.

INTRODUCTION

The advent of waveform (graphic) analysis marked the beginning of a new and exciting era in ventilator-patient management for respiratory care professionals. Waveforms give us the capacity to observe and document real-time measurements of patient-ventilator interactions. In the past, many problematic interactions between the patient and ventilator that were suspected could not be confirmed without sophisticated equipment and time-consuming effort. Now, someone skilled at analyzing waveforms can evaluate patient-ventilator synchrony, ventilator function, pulmonary status, and appropriateness of ventilator adjustments in a matter of seconds. Also, hard copies of graphics depicting improvements in pulmonary function, ventilator management, and respiratory care can be documented. It should be common practice for practitioners to use waveforms to assist in ventilator-patient assessment and management.

To develop expertise in waveform analysis requires an in-depth understanding of the principles that govern the shape of waveforms and the characteristics of the scalars measured: flow, pressure, and volume over time, and the dynamics of the ventilator-patient interface. Skill is enhanced through mathematical analysis and laboratory exercises. Prior to clinical practice, it is essential to analyze graphics during simulations of different patient-ventilator interactions in the laboratory. Test lungs should be used to simulate changing airflow resistance, compliance and I:E ratios; and to create conditions such as gas leak, air trapping, and auto-PEEP.

In clinics, graphics should always be displayed during mechanical ventilation and analyzed before and after implementing ventilator adjustments and therapy. This chapter provides the students and clinicians the basic knowledge to improve ventilator-patient management using waveform analysis. Refer to Table 11-1 as a reference for key abbreviations used within this chapter.

TABLE 11-1 Key Abbreviations

C_{LT}	Lung-thorax compliance (static compliance)
CFW	Constant flow waveform
CMV	Controlled mandatory ventilation
DRFW	Descending ramp flow waveform
FVL	Flow-volume loop
IRPCV	Inverse ratio pressure-controlled ventilation
P_{ALV}	Alveolar pressure
P_{AO}	Airway opening pressure
P_{TA}	Transairway pressure
PCV	Pressure-controlled ventilation
Peak P_{ALV}	Peak alveolar pressure; plateau pressure
PIP	Peak inspiratory pressure
PVL	Pressure-volume loop
SIMV	Synchronized intermittent mandatory ventilation
T_E	Expiratory time
T_I	Inspiratory time
TCT	Total cycle time
VCV	Volume-controlled ventilation
V_T	Tidal volume

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FLOW WAVEFORMS DURING POSITIVE PRESSURE VENTILATION

Flow, pressure, and volume are the three variables measured and displayed by graphics in real time. **Pressure-volume loops (PVLs)** and **flow-volume loops (FVLs)** are also available. As shown in Figure 11-1, depending on conditions, modes, and manufacturers, six distinct flow patterns can be set or can develop during positive-pressure ventilation (PPV): the **constant flow waveform (CFW)**; the convex rise (dashed line) in flow; the descending ramp or concave pattern (dashed line); the ascending ramp, and sine flow patterns. The CFW can present a convex pattern (dashed line) if the rise time to peak flow rate is slowed for patient comfort during **volume-controlled ventilation (VCV)**. What is commonly called the decelerating flow waveform is more appropriately called a **descending ramp flow waveform (DRFW)** (Chatburn, 2001, 2007). Depending on the manufacturer, a ventilator may offer a "true" DRFW that descends from the initial peak flow level to zero-end-flow as presented in Figure 11-1, or one that descends to some preset end-flow level above baseline. During **pressure-controlled ventilation (PCV)**, a DRFW may present an exponential decay or concave pattern (dashed line) depending on lung characteristics and patient effort.

The ascending ramp and sine (also called sinusoidal) waveforms are seldom used or available for PPV because the initial flow rate is not sufficient to accommodate synchronized assisted ventilation for most patients. The fast rise to peak flow offered by the CFW and DRFW patterns has proven to be superior in meeting patient flow demands in clinics and in research.

pressure-volume loop (PVL): Graphic display of changes in pressure and volume during a complete respiratory cycle.

flow-volume loop (FVL): Graphic display of changes in flow and volume during a complete respiratory cycle.

constant flow waveform (CFW): Flow-time waveform where the peak flow occurs at or near beginning inspiration and remains constant until end-inspiration.

volume-controlled ventilation (VCV): Mechanical ventilation that allows the RCP to set the mandatory tidal volume.

descending ramp flow waveform (DRFW): Flow-time waveform where the peak flow occurs at or near beginning inspiration and decreases to baseline at end-inspiration.

pressure-controlled ventilation (PCV): A mode of ventilation in which the peak inspiratory pressure is preset and remains stable in conditions of changing compliance and airflow resistance.

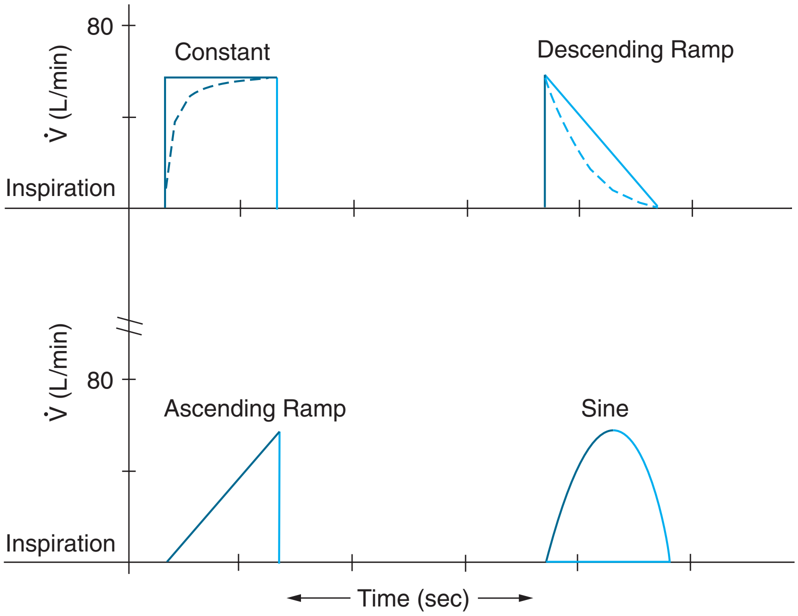

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FIGURE 11-1 Six flow waveforms available for positive pressure ventilation: constant flow, convex constant flow pattern (dotted line), descending ramp, concave descending ramp pattern (dotted line), ascending ramp, and sine flow pattern.

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The use of the sine or ascending ramp flow waveforms may be appropriate for controlled ventilation where patient effort, flow, or volume of gas being demanded is not an issue. When a patient is heavily sedated and there is no patient effort to breathe, the slow rise to set peak flow levels may improve lung gas distribution because there is less resistance to gas flow. Higher flow rates cause higher resistance to flow. Also, when there is variable flow resistance in diseased airways throughout the lungs, gas follows the path of least resistance, preferentially ventilating normal lung parenchyma. Utilizing slower flow rates or rise time to set peak flow levels may reduce flow resistance and improve gas distribution to the poorly ventilated areas of the lung. During assisted (patient-triggered) ventilation, however, there is a time lag between patient demand for flow because of ventilator inspiratory valve opening response time and time for gas to accelerate to the flow level demanded. When the initial flow level is set higher than demanded, it will often compensate for this time lag and improve ventilator-patient synchrony (Marini et al., 1985).

EFFECTS OF CONSTANT FLOW DURING VOLUME-CONTROLLED VENTILATION

Figure 11-2 displays two theoretical sets of graphics or waveforms of volume-controlled, ventilator-initiated breaths: a set of ideal flow (top) and pressure (bottom) waveforms on the *y*-axis that are contiguous in time (*x*-axis), followed by a second

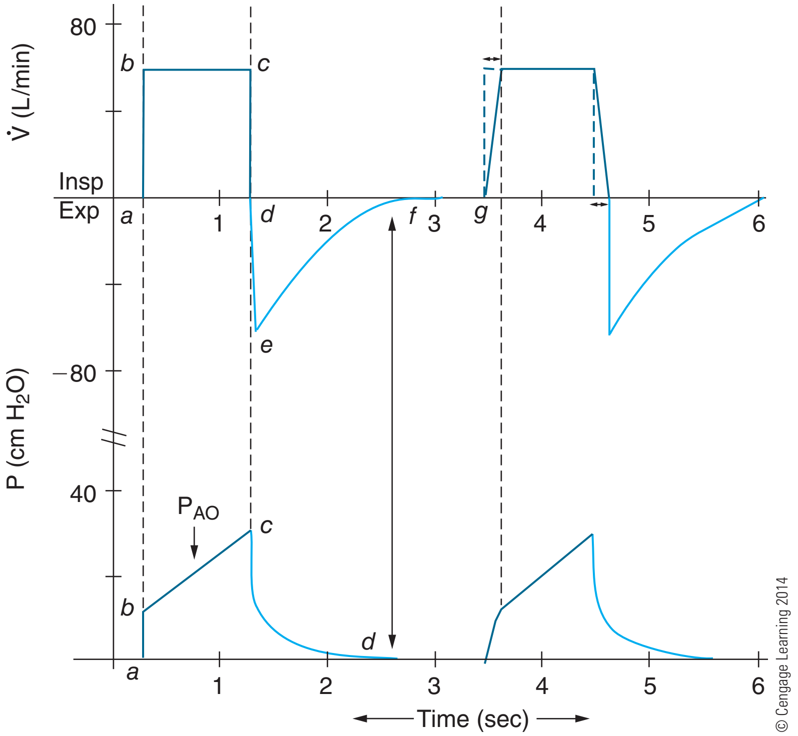

FIGURE 11-2 Two sets of flow- and pressure-time waveforms. The letters in the first set (left - $\dot{V}$ and P) mark the various phases of the respiratory cycle (see text). The second set (right - $\dot{V}$ and P) shows a delay in rise time to peak flow.

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set of waveforms of the same type, but with characteristic changes made for comparison. Both sets of waveforms represent mandatory volume-controlled breaths or volume-controlled ventilation (VCV). Inspiration in these examples of VCV is begun or triggered by the ventilator. All breaths during VCV are volume- or flow-controlled, and ended (cycled into expiration) by the ventilator. These are also considered examples of volume-controlled ventilation. When a targeted value reached, such as the volume set, is used to cycle the ventilator into expiration, the parameter or variable targeted is considered to be limited. And the mode targeting that variable to cycle the ventilator into expiration can be characterized as limited as well as controlled. The letters in the graphics represent the various components and phase variables of a breath recorded by flow-and pressure-time graphics (Chatburn, 2001, 2007).

Flow-Time Waveform

In the first flow-time waveform (Figure 11-2, left - $\dot{V}$ and P) the letters represent the four phases of the ventilatory or respiratory cycle, the period of time from the beginning of one breath and the beginning of the next. The letter *a* presents the end of expiration and the beginning of the inspiration where flow is ventilator- or time-triggered. It is always a positive upward stroke on ventilator graphics. Letter *b* marks the inspiration with a peak and constant flow of 60 L/min.

Letter *c* marks the change from inspiration to expiration where the breath is volume- or time-cycled into expiration. The inspiratory flow waveforms represent conceptual or idealized waveforms. The initial flow cannot reach the peak flow level instantaneously. No ventilator can perfectly "square off" flow and pressure waveforms as they are presented in textbooks, or by ventilator graphic software. Realistic waveforms will have more rounded or slightly jagged corners and variable patterns (so-called noise) with transitive changes in flow and pressure as inspiratory and expiratory valves make rapid adjustments in flow rates. Noise, however, is mitigated by reducing the number of data sampled (measured) per second and digitized by the ventilator's hardware for graphic presentation. Approximately 30 to 50 samples of flow or pressure measurements are digitized per second, which creates smoother-appearing lines, slopes, and curves for graphic representation of waveforms. Higher sampling rates would be costly. Greater attention to minor fluctuations in measurements and details is not necessary clinically, nor for graphic presentations in textbooks, to learn the concepts and major principles involved with waveform analysis. Thus, minor details to graphics have been omitted for ease of presentation and mathematical analysis. Clinically relevant exceptions may be presented and explained.

Letter *d* depicts expiration, the fourth phase of the ventilatory cycle, which is always to the lower side of baseline or zero flow. Letter *e* represents the peak expiratory flow rate attained (60 L/min), which is assigned a negative value in graphics. The expiratory flow pattern from the peak level attained to the end of flow is normally an exponential decay and convex pattern under passive conditions. Letter *f* represents the end of a patient's flow as it returns to baseline, and *g* is the passive expiratory pause time in flow until the next breath.

Pressure-Time Waveform

The ideal pressure-time waveform that is created under passive conditions of constant flow ventilation is a step ascending ramp. Letter *a* on the pressure-time waveform in Figure 11-2 indicates the beginning of inspiration and corresponds in time to the flow waveform as indicated by the dashed line connecting the two waveforms. The beginning of the pressure waveform provides information about the triggering variable of the inspiratory phase of ventilation. There is no patient effort or assist breath (see Figure 11-27 for an example of an assist breath), which indicates that the initial flow from the ventilator is time-triggered. The initial flow pushes gas from the ventilator circuit into the patient's lungs as it accelerates to peak flow level. Little volume is actually delivered to the lungs during this initial time period. Only a few milliliters of gas need to pass through the pneumotachometer at the beginning of the inspiratory limb of the circuit for the peak flow rate to be attained. In addition, some gas volume is lost (not delivered to the patient's lungs) as pressure rises in the circuit because of large bore tubing compliance (tubing expansion) and compression of gas molecules— resulting in a higher density or gas per area and gas being "lost" in the circuit during inspiration and not being delivered to the patient's lungs. Approximately 2 to 3 mL/cm H_2O of compressible volume lost is common for disposable adult circuits unless the ventilator offers volume compensation (MacIntyre et al., 2008).

The initial rise in pressure (the vertical step prior to the linear rise in pressure) is mostly the result of resistance to flow through the ventilator circuit and endotracheal tube (Tobin, 1994). The back pressure resulting from impedance to ventilation (flow resistive pressure caused by tubing and airways, and lung tissue recoil pressure) is graphically recorded by a manometer at the inspiratory valve of the ventilator. Letter *b* represents the change in slope on a pressure waveform that occurs once peak flow is reached. Then, the peak flow level is sustained (constant) throughout inspiration. Once flow delivery from the ventilator becomes constant, there is a relatively linear rise in the dynamic or **airway opening pressure (P_{AO})**, which closely parallels the linear rise in **alveolar pressure (P_{ALV})** until the **peak inspiratory pressure (PIP)** and peak P_{ALV} are reached at end-inspiration.

airway opening pressure (P_{AO}): Sum of transairway pressure (P_{TA}) and alveolar pressure (P_{ALV}).

alveolar pressure (P_{ALV}): Pressure required to overcome the elastic recoil property of the lungs.

peak inspiratory pressure (PIP): Highest pressure during the inspiratory phase.

Flow cannot be constant at both ends of a ventilator circuit, however, because volume is lost per unit rise in pressure as a result of tubing compliance (expansion) and gas compression. A loss in volume to the ventilator circuit equates to a loss in flow (flow = volume per time) to the lungs. There has to be some reduction in flow from the ventilator end of the circuit when compared to the flow through the patient's lungs at the other end of the circuit (carina). Flow is constant on graphics as it leaves the ventilator. The reduction in flow from beginning to end-inspiration depends on the characteristics of compliance and airflow resistance.

Letter *c* marks the PIP, the end of inspiration, and the beginning of expiration where the ventilator is time- and volume-cycled into expiration. The second dashed line shows that the end of inspiratory flow and PIP are contiguous in time. As flow exits through the expiratory limb of the circuit, pressure is created by the resistance to flow through the circuit and measured by a manometer at the expiratory valve. Pressure subsides as gas is released into the atmosphere. Letter *d* marks

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the end of expiratory pressure being sensed by the pressure manometer. The bold, double-headed arrow shows that flow is still being recorded as gas is passing through the pneumotachometer at the expiratory valve. The manometer is not sufficiently sensitive or appropriately placed at the expiratory valve for pressure to be continuously recorded until end-expiratory flow is reached on some ventilators.

In the second flow waveform (Figure 11-2), the time involved (upper double-headed arrow) for the rise to peak flow is exaggerated to show a time lag between the beginning of inspiration and attainment of peak flow. This example is presented only to evaluate the idea conceptually. This delay in time corresponds to a loss in flow, and, therefore, of volume in the initial phase of inspiration. At the end of inspiration, the volume lost initially is regained as the flow slows to zero over the same delay in time and area under the flow pattern (lower doubled-headed arrow). The peak flow could have been increased to regain the volume lost initially, instead of time being extended. Thus, flow-controlled ventilation equates to volume-controlled ventilation. The concept concerning volume and the area under the flow pattern is explained in more detail in Figure 11-3. The dashed line from peak flow to the recorded pressure demonstrates that once peak flow is reached and flow remains constant, the initial rise in predominantly flow-resistive pressure changes slope and rises linearly to PIP, as in the preceding example. The slope of the initial rise in pressure is lower than in the preceding vertical example because of the slower rise to peak flow.

✓ (Figure 11-3) Area *a* (inspired volume) = Area *b* (expired volume). If volume under area *b* is less than volume under area *a*, air leak or air trapping may be present.

The basic mathematical principle demonstrated in Figure 11-3 is that area, under a flow-time curve or pattern, equals volume. We know from geometry that the area of a rectangle equals length times width (A = L × W). On the flow graph, the length

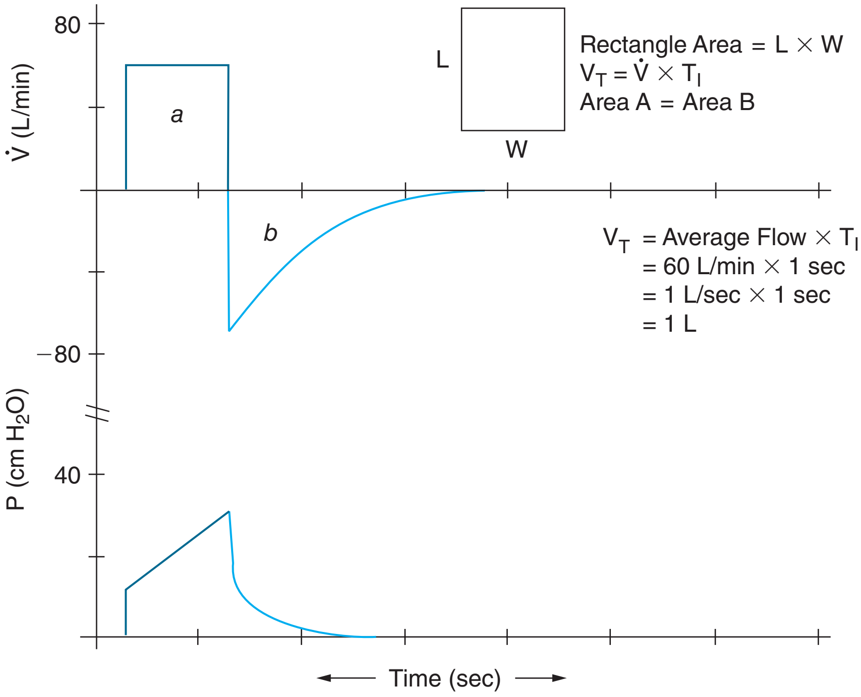

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FIGURE 11-3 Flow waveform to show that area under a constant flow curve equals volume. Tidal volume is the product of constant flow and inspiratory time: V_T = Flow × T_I.

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tidal volume (V_T): Volume delivered by ventilator during mandatory breaths.

✓ Constant Peak Flow × I Time = Tidal Volume

side of the waveform (*y*-axis) is 60 L/min or 1 L/s (60 L/min × 1 min/60s = 1 L/s). Flow is constant for 1 second, therefore, the width of the rectangle (*x*-axis) is 1 second and, since length times width equals area, 1 L/s × 1s = 1 liter **tidal volume (V_T)** delivered. Also, constant flow means there is a constant delivery of volume per unit time. For example, if 0.25s is used as the unit of time, a 0.25 liter of gas is delivered every 0.25s (Volume = 1 L/s × 0.25s = 0.25 L).

Since patients exhale what they inhale, it can be stated that area *b* enclosed under the expiratory flow wave equals area *a* under the CFW. If the volume enclosed by area *b* during mechanical ventilation is less than area *a*, then there must either be a leak in the circuit, some gas has not been expired, or has been trapped in the patient's lungs momentarily. The pressure pattern shows a constant rise in lung pressure during the constant flow period as discussed earlier in Figure 11-2.

inspiratory time T_I: Time from beginning inspiration to end of inspiration and beginning expiration.

Figure 11-4 depicts the ideal pressure waveform with details that correspond to the enclosed square flow wave presented above it. In this example, a 0.5 s pause in delivery of flow from the ventilator has been set (prolonging **inspiratory time T_I**). The pause in flow delivery results in a static pressure measurement being maintained at the same level for 0.5 sec, creating a plateau or pause pressure at the end of the waveform.

For the flow waveform, the double-headed arrow shows that no flow is being delivered from the ventilator for 0.5 s. During this time period, the inspiratory and expiratory valves of the ventilator are closed to hold gas volume constant in the

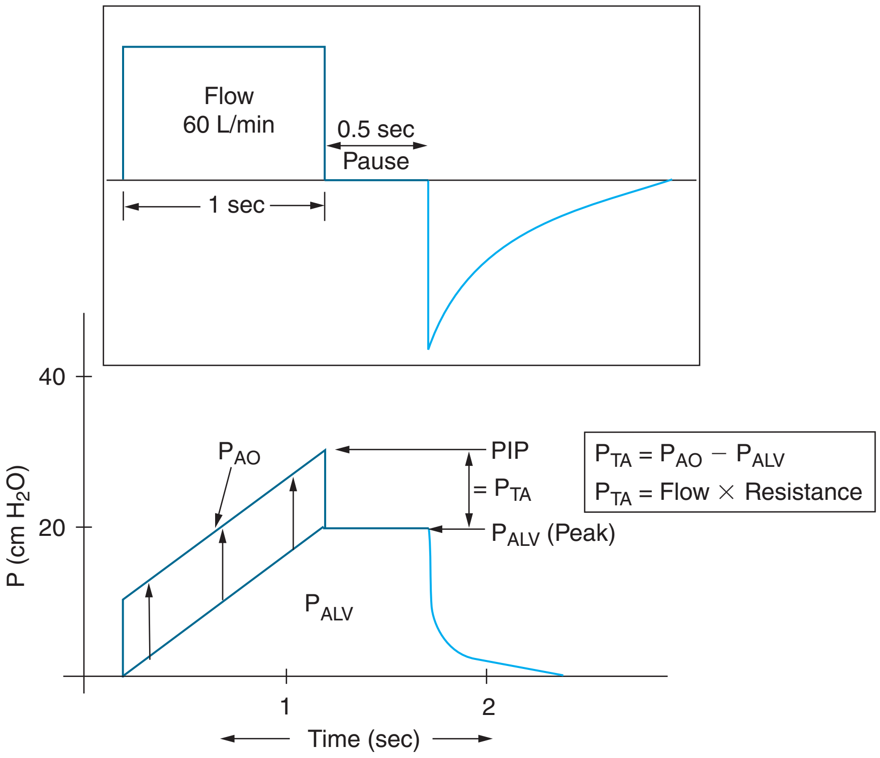

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FIGURE 11-4 Use of end-inspiratory pause to create and measure peak alveolar pressure (peak P_{ALV} or plateau pressure) and transairway pressure (P_{TA}). Peak P_{ALV} or plateau pressure is used to calculate static compliance; P_{TA} is used to calculate airflow resistance.

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patient's lungs, allowing clinicians to measure lung pressure or, more importantly, alveolar pressure (P_{ALV}). Since there is no flow, the corresponding pressure created by resistance to flow dissipates immediately. Pressure drops to the **peak alveolar pressure (P_{ALV}), plateau pressure (P_{PLAT})** level, which can be measured because of the open communication in the ventilator circuit between the alveoli and the manometer at the ventilator. All lung pressures, not just peak P_{ALV}, are monitored during this pause in flow. However, P_{ALV} is used in the graphics and text for emphasis throughout this chapter. The extremely thin type 1 pneumocytes (0.5 to 2 angstroms) lining alveoli are more sensitive to pressure and trauma than the progressively thicker airways. P_{ALV} is a major concern and reason for performing the pause-pressure measurement during ventilator management. Once peak P_{ALV} is known, circuit and airway resistance can be determined [Resistance = (PIP − peak P_{ALV})/Flow], provided that a constant flow pattern is present to cause a consistent flow-resistive pressure throughout inspiration, as shown in Figure 11-4.

peak alveolar pressure (P_{ALV}): Pressure obtained by performing an end-inspiratory pause, also called plateau pressure.

plateau pressure (P_{PLAT}): Pressure obtained by performing an end-inspiratory pause, also called peak alveolar pressure.

✓ (Figure 11-4)
$P_{AO} = P_{TA} + P_{ALV}$

Technically, calculation of circuit and airway resistance should be the pressure gradient between PIP and peak P_{ALV}. Pause pressure (P_{pause}) is intended to eliminate the contribution of the resistance to flow through the airways, and it is synonymous with peak P_{ALV} in the equation for circuit and airway resistance measurements practiced clinically. Measuring P_{pause} enables calculation of the **lung-thorax compliance (C_{LT})** (e.g., C_{LT} = volume/pressure, and observing the graphic it can be determined that $C_{LT} = 1\ L/20\ cm\ H_2O = 0.50\ L/cm\ H_2O$). The pressure-time waveform in Figure 11-4 shows, as noted earlier, that the P_{AO} represents two distinct pressures involved during inspiration with flow: the pressure caused by resistance to flow through the circuit and airways, and the elastic recoil pressure created by the airways, alveoli (P_{ALV}), and chest wall.

lung-thorax compliance (C_{LT}): The relationship of volume and pressure (V/P) that is imposed by the property of the lungs and thorax. Also called static compliance.

✓ (Figure 11-4) At end-inspiration, P_{TA} = PIP − Peak P_{ALV} during constant flow ventilation.

Under ideal conditions, there is a linear rise in P_{ALV} (dashed line) during the inspiratory cycle since there is a constant rise in volume per unit time with constant flow delivery. Flow-resistive pressure (arrows above dashed line) created by the ventilator circuit and airways is also constant, assuming that flow through the respiratory system is constant. Flow-resistive pressure will rise parallel to the rise in P_{ALV} during constant flow. The P_{AO} is the dynamic pressure recorded by the pressure manometer that clinicians observe at the ventilator as gas is being forced into lungs. Another term for flow-resistive pressure during inspiration is **transairway pressure (P_{TA})**, which is the difference between P_{AO} and P_{ALV} ($P_{TA} = P_{AO} - P_{ALV}$). Thus, the P_{AO} is equal to the summation of the two distinct pressures during inspiration: P_{TA} and P_{ALV}. At end-inspiration, P_{TA} equals the difference between the PIP and the peak P_{ALV} (P_{TA} = PIP − Peak P_{ALV}) during constant-flow ventilation. Also, P_{TA} equals flow times resistance (P_{TA} = Flow × Resistance).

transairway pressure (P_{TA}): Flow-resistive pressure, the difference between airway opening pressure (P_{AO}) and alveolar pressure (P_{ALV}), or $P_{TA} = P_{AO} - P_{ALV}$.

✓ Airflow Resistance = P_{TA}/Flow

Controlled Mandatory Ventilation

Figure 11-5 is a waveform example of **controlled mandatory ventilation (CMV)**. CMV is a mode that defines the specific control, phase, and conditional variables of mandatory breaths (Chatburn, 2001, 2007). The CMV waveforms demonstrate that each breath is a time-triggered mandatory breath, and volume-controlled

controlled mandatory ventilation (CMV): Time-triggered mandatory breaths provided by the ventilator. Also called controlled mechanical ventilation or continuous mandatory ventilation.

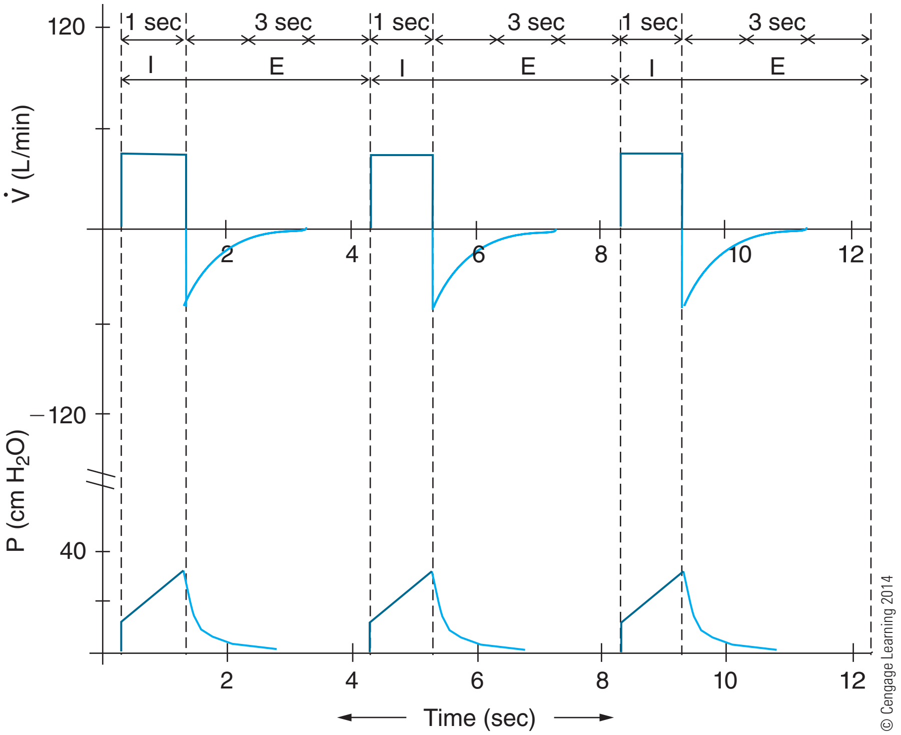

FIGURE 11-5 Flow- and pressure-time waveforms to show controlled mechanical ventilation. Note the absence of negative triggering pressure before the beginning of inspiration.

because the flow pattern is the same each breath. Today's ventilators control flow (flow controllers), and volume is indirectly controlled. Thus, the mode is considered VCV, which means that volume and flow are held constant, and pressure is allowed to fluctuate depending on the compliance and airflow resistance.

✔ Total Cycle Time (TCT) = Inspiratory time (T_I) + Expiratory time (T_E)

expiratory time (T_E): Time period from beginning expiration to beginning inspiration of next mechanical or spontaneous breath.

✔ Frequency (f) = 60 sec/TCT

As shown in Figure 11-5, the inspiratory time (T_I) is 1 sec and the **expiratory time (T_E)** is 3 sec. (Note: E time is measured from the beginning of expiration to the beginning of the next inspiration.) The total respiratory cycle time is therefore 4 sec ($TCT = T_I + T_E$). Since the TCT is 4 sec under control mode, the frequency (f) is therefore 15/min (f = 60 sec/TCT = 60 sec/4 sec = 15). The inspiratory-to-expiratory (I:E) ratio is 1:3 (1 sec:3 sec).

Assist Mandatory Volume-Controlled Ventilation

Figure 11-6 shows optimal response for two assist mandatory breaths given the same settings for the CMV mode VCV. Each pressure-time waveform demonstrates that negative pressure [−2 cm H_2O (dashed line)] is created in the ventilator circuit and graphically recorded when the patient makes an effort to inspire. The dashed line connecting the first set of waveforms shows that the inspiratory flow begins as soon as the negative deflection reaches the sensitivity threshold of −2 cm H_2O set on the ventilator. When the ventilator response to patient effort is optimal, each breath is very similar to a controlled breath, with the exception that there is a slight reduction (not noticed) in the P_{AO} during inspiration as the patient actively inspires in response to gas being forced into the lungs during assisted ventilation (Dick et al., 1996).

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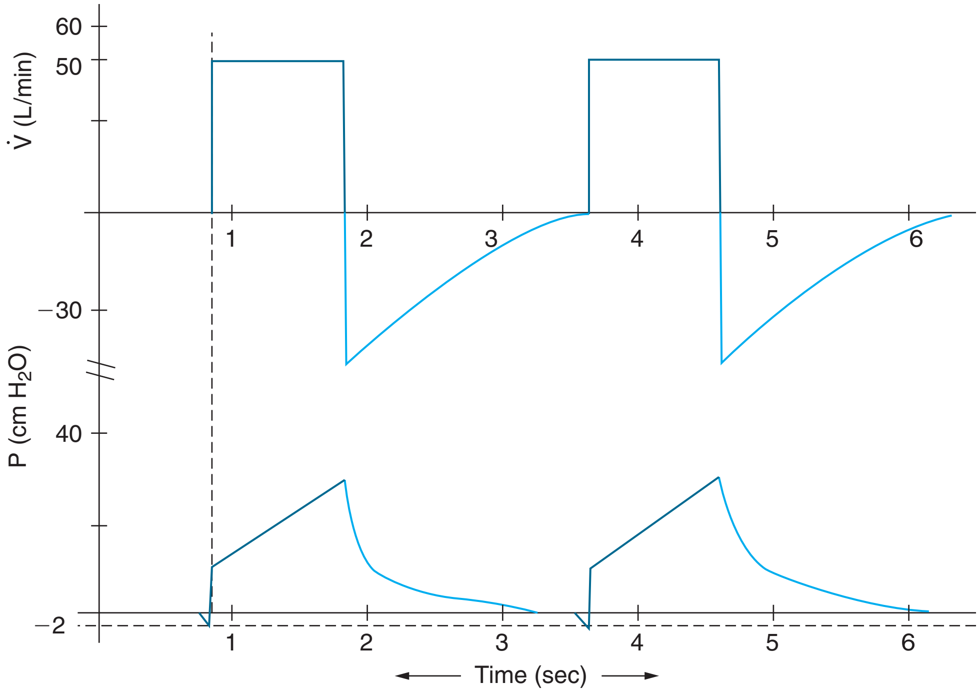

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FIGURE 11-6 Flow- and pressure-time waveforms to show assist mechanical ventilation. Note the negative triggering pressure (−2 cm H_2O) before the beginning of inspiration.

✔ (Figure 11-6) Inspiratory flow begins as soon as the negative deflection reaches the preset sensitivity of −2 cm H_2O.

✔ (Figure 11-7) The T_I is unchanged during VCV because V_T and average flow are held constant ($T_I = V_T$/Average Flow). I:E ratios differ because of changing E time.

When patients expand their thorax in synchrony with gas delivery from the ventilator, this relieves some of the inspiratory pressure that would build up in the circuit and lungs compared to a passive patient (sedated and paralyzed). Slight reductions (2 to 3 cm H_2O) in the P_{AO} pattern that are made apparent by the variability in PIP at end-inspiration are unremarkable. Patients commonly perform 33% to 50% of normal workloads of ventilation during optimal assist CMV VCV breaths (Marini et al., 1985).

Figure 11-7 demonstrates the CMV mode of ventilation again. The settings are the same as in the previous examples, except that volume waveforms have been added, PEEP is being administered at 10 cm H_2O, and the patient is receiving two assist and two mandatory breaths. For a CFW, volume rises linearly over time, as demonstrated. During expiration, volume demonstrates an exponential decay pattern in correspondence with the expiratory flow pattern. The I:E ratios are 1:3 for the second and third mechanical breaths, as the patient expires and relaxes after the second assist breath and allows the ventilator to take control (mandatory breaths). The expiratory time for the first breath is reduced because the patient expires faster and triggers a second breath (assist) prior to a timed mandatory breath taking place as indicated on the second pressure waveform. Thus, the I:E ratio (1:3) is reduced, because the patient triggers the second breath before the 3 sec expiratory time (T_E) has elapsed. The T_I is unchanged during VCV because V_T and average flow are held constant ($T_I = V_T$/Average Flow).

In the CMV mode, flow, V_T, and mandatory frequency are set, but the patient is allowed to trigger as many breaths as desired. When patients are able to trigger breaths in the CMV mode, the term *assist/control mode* is commonly used, but it is no longer recommended because it does not define a unique mode. When setting CMV, assist

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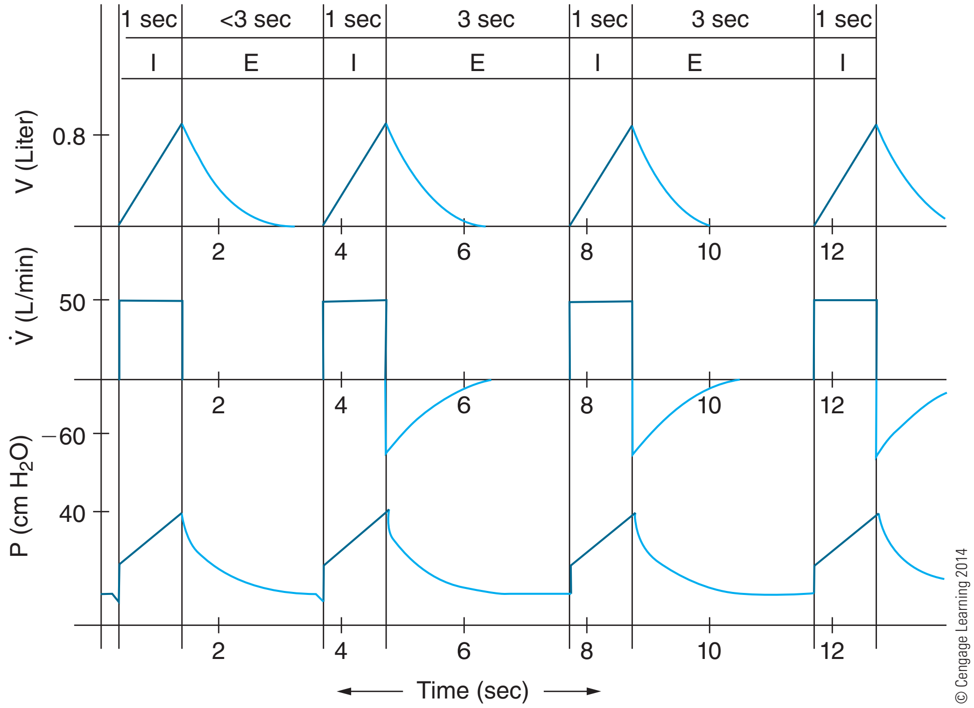

FIGURE 11-7 Volume-, flow-, and pressure-time waveforms to show two assist (first and second) and two controlled mechanical breaths (third and fourth). The pressure-time waveforms show 10 cm H_2O of PEEP. The sensitivity setting is about 2 cm H_2O below the PEEP level (first and second breaths).

mandatory breaths are set unless the sensitivity is set so low that the patient cannot reach the threshold level, which is contraindicated. Inspiratory efforts required to initiate a breath should be supported by an appropriate sensitivity setting.

The PEEP for the pressure-time waveforms is 10 cm H_2O, and since there was no change in static compliance, the PIP was increased by 10 cm H_2O as well, when compared to Figure 11-6. For the assist breaths, it can be seen that pressure drops to 8 or 2 cm H_2O below PEEP level to trigger the first two breaths because sensitivity is set at −2 cm H_2O below baseline pressure, which is known as PEEP compensation.

Mathematical Analysis of Constant-Flow Ventilation

For better comprehension of the waveforms during mechanical ventilation, respiratory care practitioners (RCPs) should be able to analyze waveforms mathematically to understand the principles involved. Figure 11-4 shows that once peak P_{ALV} for the pressure waveform is known, the P_{AO} can be delineated into the P_{TA} and P_{ALV} that develops throughout inspiration. Clinicians should be able to demonstrate mathematically that given a constant flow, the P_{ALV} and P_{AO} should rise linearly in parallel and that the P_{TA} remains constant. Given no change in lung-thorax compliance (C_{LT}), a straight line drawn from zero pressure at the beginning of inspiration to the peak P_{ALV} at the beginning of the set pause pressure should closely predict the rise in P_{ALV} throughout inspiration during constant-flow, volume-controlled ventilation.

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✔ (Figure 11-8) During constant flow ventilation, the volume and pressure rise linearly with the inspiratory time.

Figure 11-8 presents one of the mandatory volume- and pressure-time waveforms presented in Figure 11-7. The computations in Table 11-2 show the volume delivered by the CFW for each 0.25 sec unit of time under the CFW, and the volume delivered for the inspiratory time elapsed throughout inspiration (1.0 sec). Table 11-3 shows the volume computations and volume waveform, which mathematically demonstrates that the volume rises linearly during constant-flow ventilation. As shown in Table 11-4, the patient's C_{LT} equals 0.04166 L/cm H_2O. As shown, once C_{LT} is known, the equation for C_{LT} can be rearranged, and P_{ALV} can be calculated and plotted for any number of time units throughout inspiration, assuming that static C_{LT} remains constant, and the volume delivered for the time periods elapsed is known.

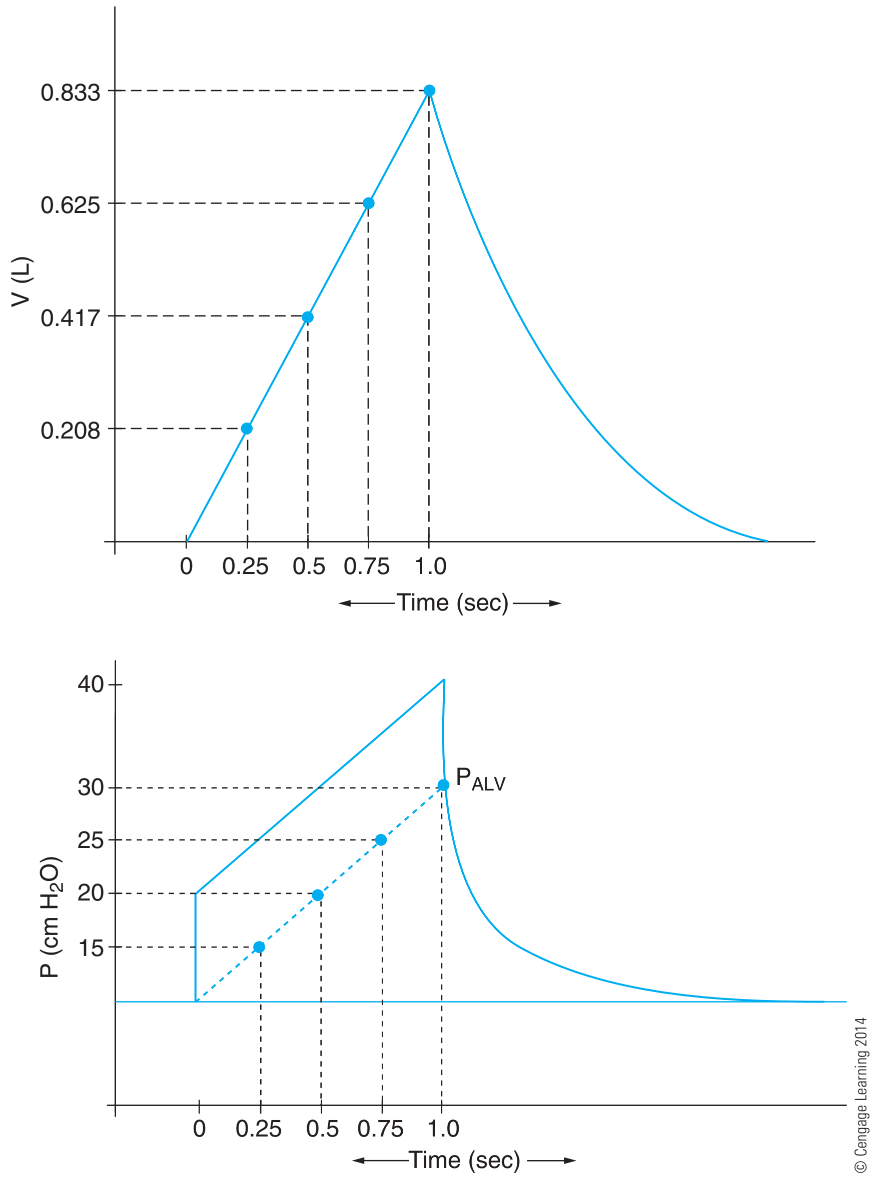

FIGURE 11-8 The volume- and pressure-time waveforms during constant flow ventilation show the steady increase of volume and pressure as inspiratory time progresses.

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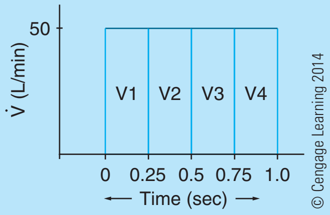
TABLE 11-2 Calculations to Determine the Volume Delivered during Constant-Flow Ventilation (See Figure 11-8)

1. Computations necessary to find volume under the CFW:

Volume = Average Flow (L/sec) × (T_I sec)

V_T = (50 L/min × 1 min/60sec) × 1.0 sec
V_T = 0.833 L/sec × 1.0 sec
V_T = 0.833 L



Volume = 0.8333 L/sec × 0.25 sec = 0.2083 L per 0.25 sec
Volume = 0.2083 L × 2 = 0.4166 L in 0.50 sec
Volume = 0.2083 L × 3 = 0.6249 L in 0.75 sec
Volume = 0.2083 L × 4 = 0.833 L in 1.00 sec

Or

Volume = 0.8333 L/sec × 0.25 sec = 0.2083 L
Volume = 0.8333 L/sec × 0.50 sec = 0.4166 L
Volume = 0.8333 L/sec × 0.75 sec = 0.6249 L
Volume = 0.8333 L/sec × 1.00 sec = 0.8333 L
V1 = 0.2083 L
V2 = 0.2083 L
V3 = 0.2083 L
V4 = 0.2083 L
V_T = V1 + V2 + V3 + V4 = 0.833 L

TABLE 11-3 Calculations to Determine the Inspiratory Volume Waveform that Develops during Constant-Flow Ventilation (See Figure 11-8)

1. Computations necessary to plot the volume pattern during inspiration:

Volume = Average Flow (L/sec) × T_I(sec)
V_T = (50 L/min × 1 min/60sec) × 1.0 sec
V_T = 0.833 L/sec × 1.0 sec
V_T = 0.833 L

Volume = 0.8333 L/sec × 0.25 sec = 0.2083 L
Volume = 0.8333 L/sec × 0.50 sec = 0.4166 L
Volume = 0.8333 L/sec × 0.75 sec = 0.6249 L
Volume = 0.8333 L/sec × 1.00 sec = 0.8333 L

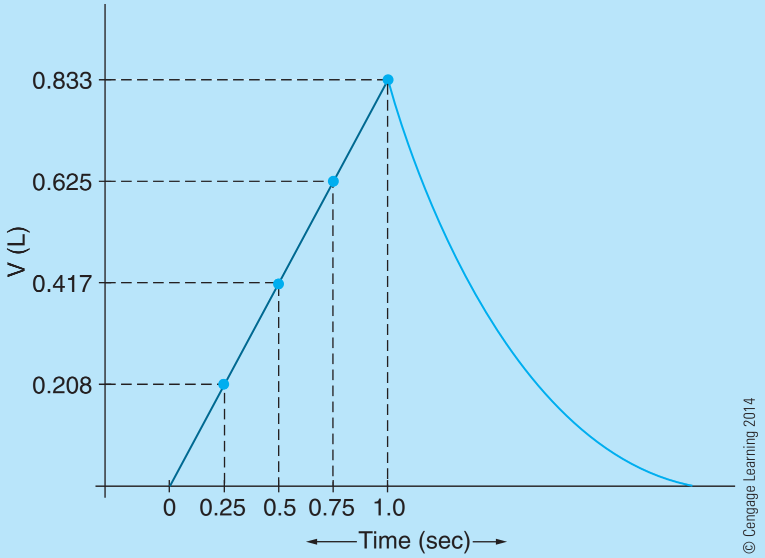


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TABLE 11-4 Calculations to Determine the P_{ALV} Pattern that Develops during Constant Flow Ventilation (See Figure 11-8)

$C_{LT} = V_T/(P_{ALV} - PEEP)$
$C_{LT} = 0.833\ L/(30\ cm\ H_2O - 10\ cm\ H_2O)$
$C_{LT} = 0.833\ L/20\ cm\ H_2O$
$C_{LT} = 0.4166\ L/cm\ H_2O$

$C_{LT} = V_T/(P_{ALV} - PEEP)$
$P_{ALV} = V_T/C_{LT} + PEEP$

$P_{ALV} = 0.8333\ L/0.04166\ L/cm\ H_2O + 10\ cm\ H_2O = 30\ cm\ H_2O$
$P_{ALV} = 0.6249\ L/0.04166\ L/cm\ H_2O + 10\ cm\ H_2O = 25\ cm\ H_2O$
$P_{ALV} = 0.4166\ L/0.04166\ L/cm\ H_2O + 10\ cm\ H_2O = 20\ cm\ H_2O$
$P_{ALV} = 0.2083\ L/0.04166\ L/cm\ H_2O + 10\ cm\ H_2O = 15\ cm\ H_2O$

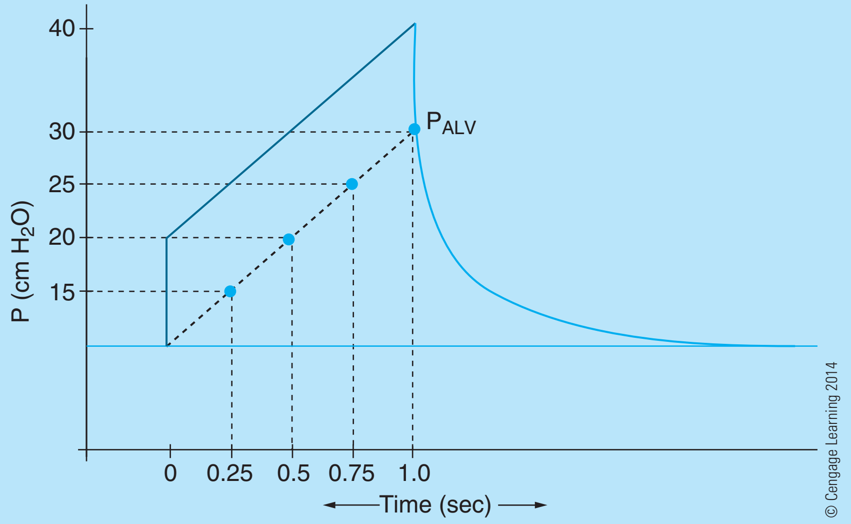

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Figure 11-8 demonstrates drawing a line (dashed line) through the values plotted for the volume delivered and the P_{ALV} developed over time. This demonstrates also that P_{ALV} rises linearly and in parallel to the P_{TA} throughout inspiration.

SPONTANEOUS VENTILATION DURING MECHANICAL VENTILATION

Synchronized Intermittent Mandatory Ventilation

synchronized intermittent mandatory ventilation (SIMV): A mode of ventilation in which mechanical breaths are delivered without overlapping spontaneous breaths.

Figure 11-9 shows the three types of breaths (controlled, spontaneous, and assist) offered during **synchronized intermittent mandatory ventilation (SIMV)**. The first set of waveforms (Figure 11-9, left) is ideal flow- and pressure-time patterns for a volume- or flow-controlled mandatory breath. Letter *a* (arrow) beneath the first pressure waveform indicates that the breath is ventilator-initiated (time-triggered and controlled).

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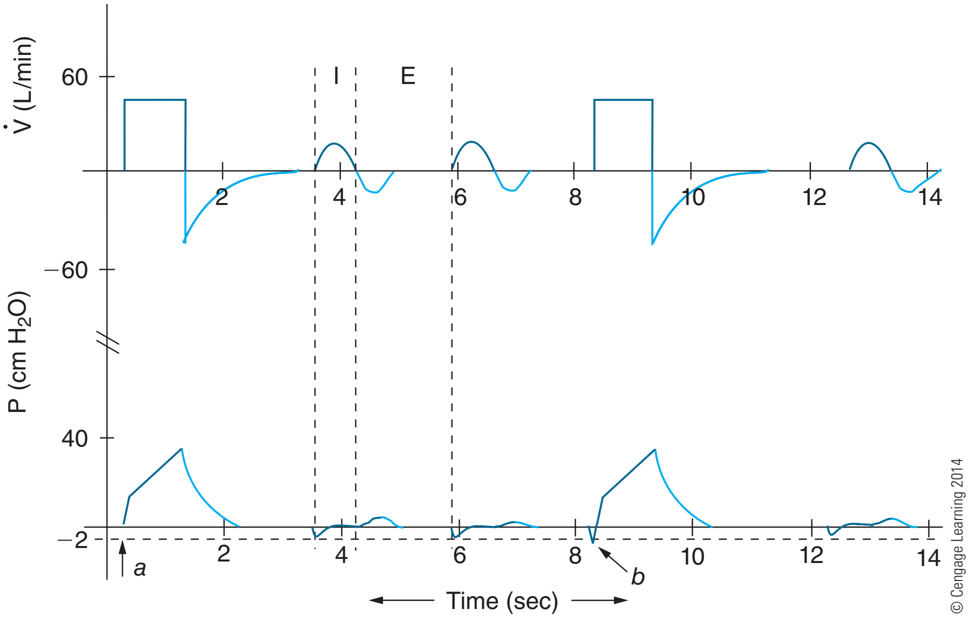

FIGURE 11-9 Synchronized intermittent mandatory ventilation (SIMV) waveform showing one controlled (time-triggered) mandatory breath (arrow *a*), one assist (patient-triggered) mandatory breath (arrow *b*), and three spontaneous breaths (second, third, and fifth breaths).

Since the breath is mandatory, it shows that the patient did not attempt to breathe within the window of time (SIMV period) set up for synchronized (assist) mechanical breaths, and a timed mandatory breath was delivered as required for the SIMV mode. Two sets of flow and pressure-time waveforms depicting ideal spontaneous breaths are recorded next during the time (spontaneous period) allotted for spontaneous breaths prior to initiation of another SIMV period by the ventilator. Given a rested patient and synchronous conditions, sine waves are created by spontaneous inspirations during the spontaneous period. As in prior examples, these waveform examples are ideal, but roughly similar to clinical presentations. Spontaneous breathing is accommodated on ventilators during SIMV and continuous positive airway pressure (CPAP) ventilation. During spontaneous ventilation, the patient controls flow, volume, inspiratory time, and expiratory time. The ventilator responds to patient effort by supplying the flow demanded. The spontaneous breaths show that the patient created a peak inspiratory flow of about 20 L/min and peak expiratory flows of about 15 L/min. The pressure waves during spontaneous breathing show a negative pressure to trigger demand flow and some positive pressure recorded in the circuit during expiration. Spontaneous breaths are explained in more detail in Figure 11-10.

The next mechanical breath in Figure 11-9 is an assist breath. Letter *b* (arrow) indicates that the patient's effort occurred to trigger the breath during the SIMV period. The manufacturer determines the amount of time allotted for synchronized mechanical and spontaneous breaths. For the next breath in the graphic, sufficient time has not elapsed for a mechanical breath to be synchronized with the patient's effort, so flow delivery is based on demand, and another spontaneous breath is provided. An SIMV rate of 6/min has probably been set for the patient in this graphic, with 5 to 6 sec allotted for the spontaneous breaths and 4 to 5 sec allotted for the SIMV breaths.

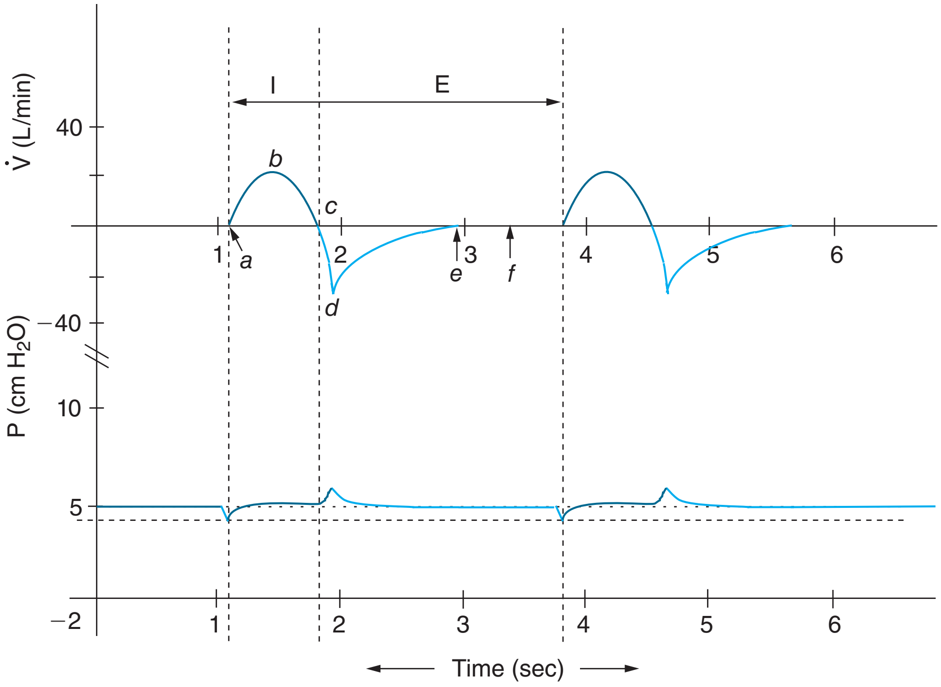

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FIGURE 11-10 Normal flow- and pressure-time waveforms during CPAP (spontaneous breathing with PEEP). The CPAP level is 5 cm H_2O.

Continuous Positive Airway Pressure

✓ (Figure 11-10) In CPAP, airway pressure is above 0 cm H_2O and all breaths are spontaneous.

✓ (Figure 11-10) In CPAP, inspiratory effort triggers and initiates flow once the negative pressure reaches the sensitivity threshold.

Figure 11-10 is a more detailed example of spontaneous ventilation during 5 cm H_2O of continuous positive airway pressure (CPAP). In the flow graphic, letter *a* (arrow) labels the end of expiration and start of inspiration; letter *b* marks the peak flow reached (20 L/min) for an idealized sine flow wave created by the patient during spontaneous breathing; letter *c* indicates the change from inspiration to expiration; letter *d* indicates the peak expiratory flow attained (20 L/min); letter *e* indicates the end of expiratory flow; and letter *f* indicates the pause period before next breath. The patient creates an inspiratory flow time of approximately 0.75 sec, an expiratory flow time of 1.25 sec, and an expiratory pause time of 0.75 sec. The dashed lines connecting the flow and pressure recordings in time delineate the total respiratory time into the inspiratory and total expiratory time periods. As with mechanical breaths, inspiratory effort (negative pressure recorded) triggers flow on demand (demand CPAP) once the negative pressure reaches the sensitivity threshold [−1 cm H_2O below CPAP level of 5 cm H_2O (dashed line)], or the pressure drops during a continuous flow CPAP. Ideally, gas is delivered to the patient according to the flow demands, which means that pressure will be maintained at baseline throughout the inspiratory phase of ventilation. In this example, the pressure recorded during inspiration is kept near baseline, indicating patient flow demand is being met. During expiration, as gas is expired into the circuit, a positive pressure is momentarily recorded until the expiratory flow is too slow or the expiratory circuit and valve resistance is so low that near-baseline pressure is recorded.

EFFECTS OF FLOW, CIRCUIT, AND LUNG CHARACTERISTICS ON PRESSURE-TIME WAVEFORMS

Flow and Transairway Pressure

✓ (Figure 11-11) Increase in flow rate causes a higher airflow resistance and thus a corresponding increase in PIP and P_{TA}. Peak alveolar pressure (peak P_{ALV}) or plateau pressure is not affected by changes in flow rate.

An end-inspiratory pause has been set in Figure 11-11 to delineate the P_{AO} into the two distinct pressures, P_{TA} and P_{ALV} (double-headed arrows), generated during gas flow through a ventilator circuit into a patient's lungs. For waveforms analysis, it is important to know how each of these pressures is affected by changes in flow and lung characteristics. Figure 11-11 demonstrates the effects of increased inspiratory flow or airflow resistance on a pressure-time waveform, which can be compared to the effects on them created by decreased compliance (Figure 11-12). The pressure-time graphics in Figure 11-11 depict (double-headed arrows) that there has been a rise in the P_{TA} from beginning to end in the second pressure waveform because of an increase in turbulence when the constant flow was doubled from 60 to 120 L/min in the second flow wave. As gas flows through a circuit system with different twists and turns, and with varied lumen sizes such as large bore tubing, humidifiers, and endotracheal tubes, turbulent flow results. In turn, the circuit system causes an

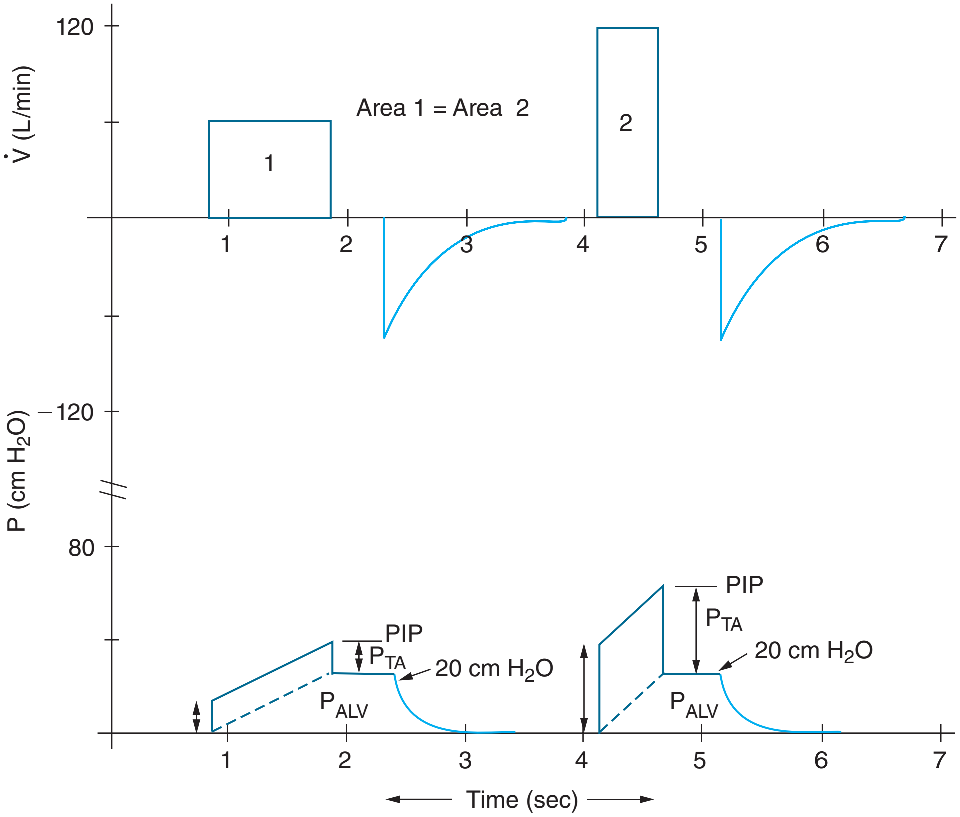

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FIGURE 11-11 Flow- and pressure-time waveforms showing the effects of flow rate on P_{TA}, P_{ALV}, and expiratory flow. Increase of inspiratory flow or airflow resistance causes an increase of P_{TA}, but no changes in peak P_{ALV} (plateau pressure) and expiratory flow.

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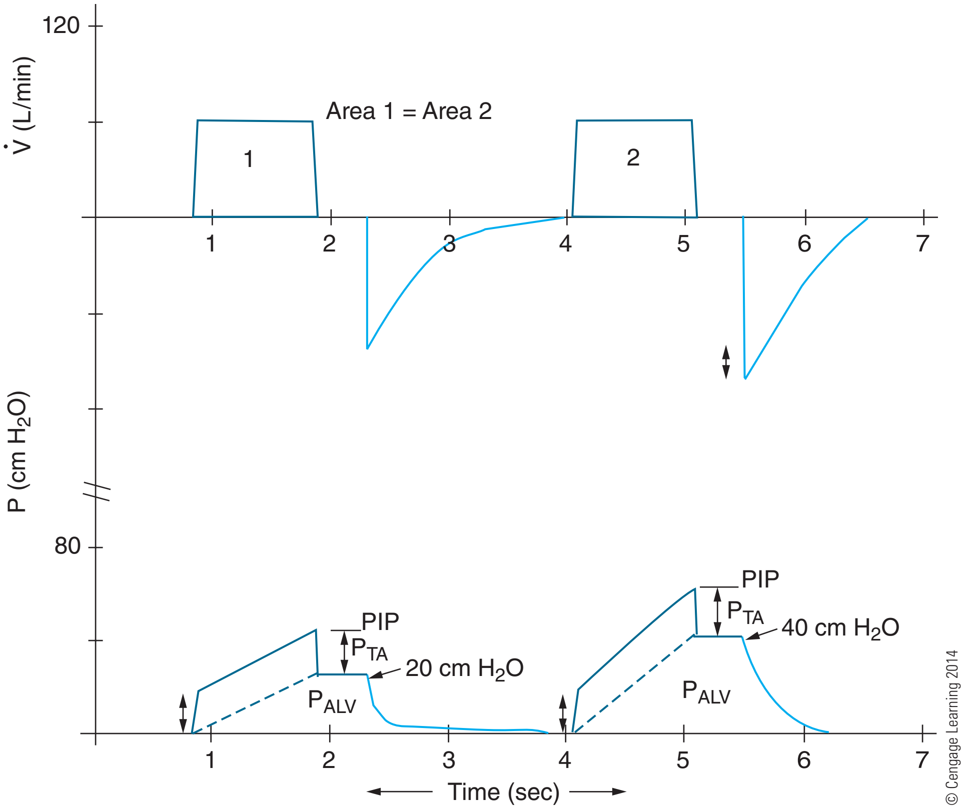

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FIGURE 11-12 Flow- and pressure-time waveforms showing the effects of lung-thorax compliance (C_{LT}) on P_{TA}, P_{ALV}, and expiratory flow. Decrease of C_{LT} causes an increase of P_{ALV} (plateau pressure) and peak expiratory flow, but no changes in P_{TA}.

exponential rise in circuit pressure with increments in flow. Conversely, an exponential decay in pressure is observed when flow is reduced (Dennsion et al., 1989). Thus, the area depicting the P_{TA} gradient ($P_{AO} - P_{ALV}$) has been more than doubled in the second pressure waveform. Note, however, that the remarkable change in P_{AO} and PIP did not affect the peak P_{ALV} because neither the C_{LT} nor the lung volume was changed. Only the rise time to peak P_{ALV} was affected because T_I was reduced by half. In this example, resistance was increased by an increase in flow. An increase in airway resistance from various lung pathologies causing bronchoconstriction and obstruction will also increase the area depicting the P_{AO} and P_{TA} gradient without a change in flow, but P_{ALV} will not be affected. Airway obstruction and bronchoconstriction will affect waveforms in other ways, which will be explained during the discussion of using expiratory waveforms as a diagnostic tool, later in this chapter.

Compliance and Alveolar Pressure

✓ (Figure 11-12) Decrease in lung-thorax compliance causes an increase in P_{ALV} and PIP. Transairway pressure (P_{TA}) is not affected by changes in compliance.

Figure 11-12 demonstrates a similar comparison between flow and pressure waveforms. PIP has substantially increased in the second example as it did in Figure 11-11. Given no change in flow or V_T to explain the increase in PIP, an end-inspiratory pause needs to be set to create the peak alveolar pressure (peak P_{ALV}), or plateau pressure for analysis. Since P_{TA} remains the same for each waveform, neither the flow nor the airway resistance has been changed. A decrease in total C_{LT} is the only explanation for the increase in PIP, which is supported by the increase in peak P_{ALV} at end-inspiration. The same peak flow, flow pattern, and volume (Area 1 = Area 2) are being used for volume delivery in each example. Total C_{LT} can be estimated by observing the information provided in the graphic comparison and, in this example, it has decreased by half. The calculated V_T under the flow curve is 1 L or 1000 mL (1 L/sec × 1 sec), and total C_{LT} is 50 mL/cm H_2O (C_{LT} = 1000 mL/20 cm H_2O) for the first example, versus 25 mL/cm H_2O (1000 mL/40 cm H_2O) in the second example.

EFFECTS OF DESCENDING RAMP FLOW WAVEFORM DURING VOLUME-CONTROLLED VENTILATION

Time- and Flow-Limited Ventilation

Figure 11-13 demonstrates the changes that occur when the flow pattern on a ventilator is adjusted from a constant to a descending ramp flow waveform (DRFW) during *time-limited* ventilation (first example) compared to *flow-limited* ventilation

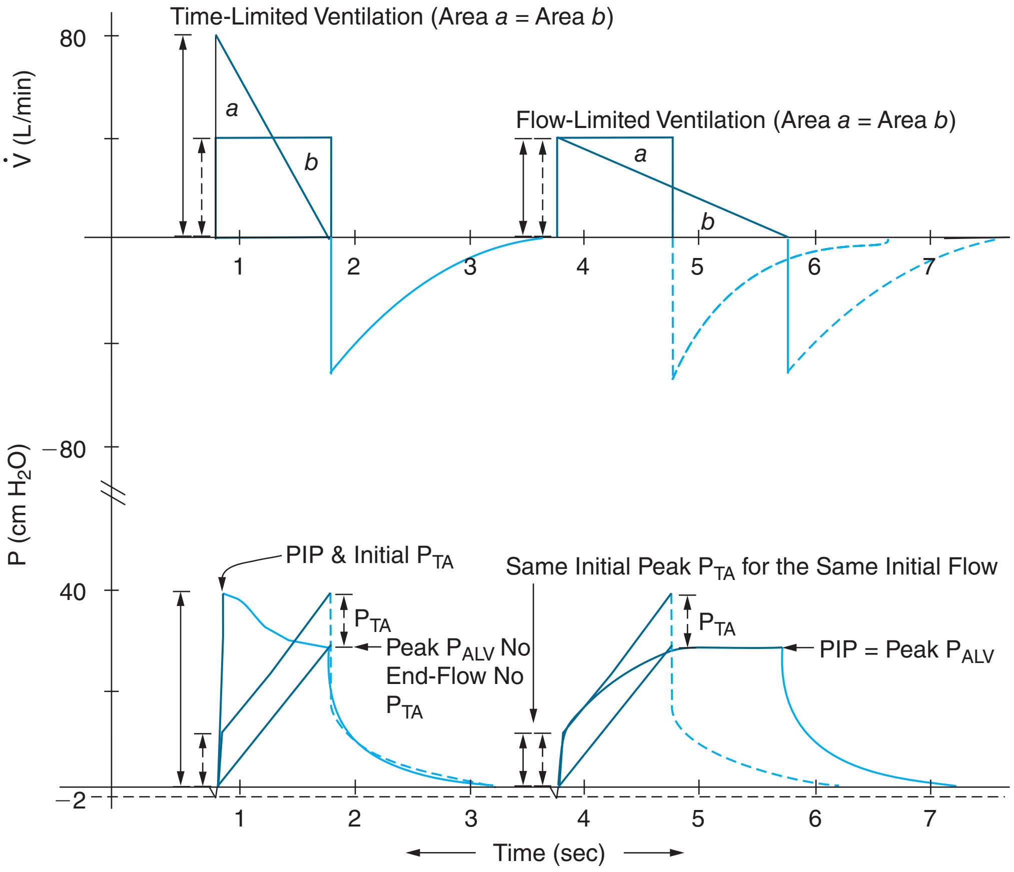

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FIGURE 11-13 Changes of dynamic pressure waveforms created by descending ramp flow during time-limited and flow-limited ventilation.

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(second example). These examples present what is known as true DRFWs (solid lines), which means that flow descends from peak level to baseline (zero-end-flow).

The type of DRFW available for selection during VCV depends on the manufacturer. For example, the Puritan Bennett 840 ventilators offer a DRFW with an end-flow of 5 L/min, and the Hamilton Medical Veolar provides the option of a true DRFW, or one with an end-flow that is 50% of the initial peak flow, called a 50% DRFW.

(Figure 11-13) During *time-limited* ventilation, the I time is unchanged when the flow pattern is changed from constant flow to descending ramp flow. The same volume can only be maintained if the *peak flow* of the descending ramp flow is increased.

"Time-limited" usage in example: T_I% is set on some ventilators. TCT and T_I% determine the I time in sec provided that the TCT and V_T remain unchanged.

The concepts presented here for Figure 11-13 are the same for the various types of DRFWs. In the first example (left), a DRFW is superimposed over a CFW (dashed lines) for comparison during time-limited ventilation, as offered by the Veolar in the CMV and SIMV modes. Inspiratory time (T_I) is held constant (time-limited) when the flow waveform selection is adjusted from a CFW to a DRFW. Since the V_T is set and T_I is held constant, the average flow rate has to remain constant (V_T = Average Flow × T_I). Average flow rate for a DRFW is 1/2 (Peak Flow + End-Flow). As demonstrated in the first comparison of flow waves example, the area enclosed by the solid and dashed line labeled *a* on the DRFW replaces the same area from the CFW area labeled *b*. For the same volume to be delivered by either flow pattern within the same time period, transition from a CFW to a true DRFW on a time-limited ventilator requires the peak flow to be doubled.

As shown in Table 11-5, the average flow rates for the CFW and DRFW are the same (0.67 L), and given the same T_I, the V_T delivered is identical. When employing a DRFW, peak flow has to be proportionately greater as end-flow is reduced if the same volume is to be delivered in the same time period.

The second example (right, Figure 11-13) of superimposed flow wave patterns demonstrates the results of switching from a CFW to a DRFW during flow-limited ventilation. The ventilator's peak flow is held constant (flow-limited) when the DRFW is selected. As the flow ramp descends, it eliminates the volume that was enclosed under area *a*. Time has to be extended until the same volume eliminated is replaced (i.e., area *b*). Given flow-limited ventilation, time fluctuates, rather than peak flow, to deliver the same V_T. Thus, depending on ventilator type, when flow is changed during CMV

TABLE 11-5 V_T Delivered by the CFW and DRFW during Time-Limited Ventilation (See Figure 11-13)

For the CFW:	For the DRFW:
PF (L/sec) = 40 L/min × min/60sec	PF (L/sec) = 80 L/min × min/60sec
PF (L/sec) = 0.67 L/sec	PF (L/sec) = 1.33 L/sec, EF = 0
V_T = Average Flow (L/sec) × T_I(sec)	V_T = Average Flow (L/sec) × T_I(sec)
V_T = 0.67 L/sec × 1 sec	V_T = ½ (PF (L/sec) + EF (L/sec) × T_I(sec)
V_T = 0.67 L	V_T = ½ (1.33 L/sec + 0 L/sec) × 1 sec
	V_T = 0.67 L

PF = Peak flow; EF = end-flow.

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TABLE 11-6 V_T Delivered by the CFW and DRFW during Flow-Limited Ventilation (See Figure 11-13)

For the CFW:	For the DRFW:
PF (L/sec) = 40 L/min × min/60sec	PF (L/sec) = 40 L/min × min/60sec
PF (L/sec) =0.67 L/sec	PF (L/sec) = 0.67, EF = 0
V_T = Average Flow (L/sec) × T_I(sec)	V_T = Average Flow (L/sec) × T_I(sec)
V_T = 0.67 L/sec × 1 sec	V_T = ½ (PF (L/sec) + EF (L/sec) × T_I (sec)
V_T = 0.67 L	V_T = ½ (0.67 L/sec + 0 L/sec) × 2 sec
	V_T = 0.67 L

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✓ (Figure 11-13) During *flow-limited* ventilation, the inspiratory peak flow is unchanged when the flow pattern is changed from constant flow to descending ramp flow. The same volume can only be maintained if the *inspiratory time* of the descending ramp flow is increased.

or SIMV from a CFW to DRFW, either the peak flow has to double (time-limited ventilation) or the T_I has to double (flow-limited ventilation) for the same V_T to be delivered. As shown in Table 11-6, since the initial flow rate for both flow waveforms is the same, the average flow rate for the DRFW is reduced by half compared to the CFW. The same V_T is delivered because T_I is doubled.

The pattern and level of pressure developed (Figure 11-13) during descending ramp flow ventilation depend, as for constant flow ventilation, on the peak- to end-flow pattern, circuit/lung resistance, and C_{LT}. As in the prior examples, the pressure waveform examples (solid lines) for the DRFWs are superimposed over the step ascending ramp pressure waveforms (dashed lines) created by the CFWs. Pressure during descending ramp flow ventilation, depending on the peak flow level set (e.g., 80 versus 40 L/min for these examples), tends to square off compared to rising linearly as it does for constant flow ventilation (see Figure 11-14). Assuming the same circuit and lung characteristics for the comparison, the higher initial peak flow for the DRFW in the first example (80 L/min) creates a higher peak flow-resistive pressure or P_{TA} (40 cm H_2O) at the beginning of inspiration on the pressure waveform, compared to flow-resistive pressure (10 cm H_2O) for the CFW. And as demonstrated, the flow-resistive pressure for DRFW decreases over time with reduction in flow, whereas the flow-resistive pressure stays constant (dashed line, double-headed arrows) for the CFW. Since the same volume is being delivered in each DRFW example, and zero flow occurs at end-inspiration, the pressure at end-inspiration for both examples is the patient's peak P_{ALV}. There is no flow at end-inspiration, so no flow-resistive pressure or P_{TA} is being created. Flow-resistive pressure steadily drops to zero during inspiration for zero-end-flow ventilation. Note that under this circumstance, the end-PIP and peak P_{ALV} are the same for the pressure waveform examples. This mechanical principle is very helpful diagnostically, because if a patient is passive during mechanical ventilation at end-inspiration, RCPs have a breath-by-breath account of the patient's peak P_{ALV} and lung compliance status!

In the pressure-time waveform on the bottom left of Figure 11-14. The PIP is at the beginning of inspiration because of the very high initial flow rate (80 L/min). During constant-flow ventilation by comparison, the PIP (dashed lines) is at the end

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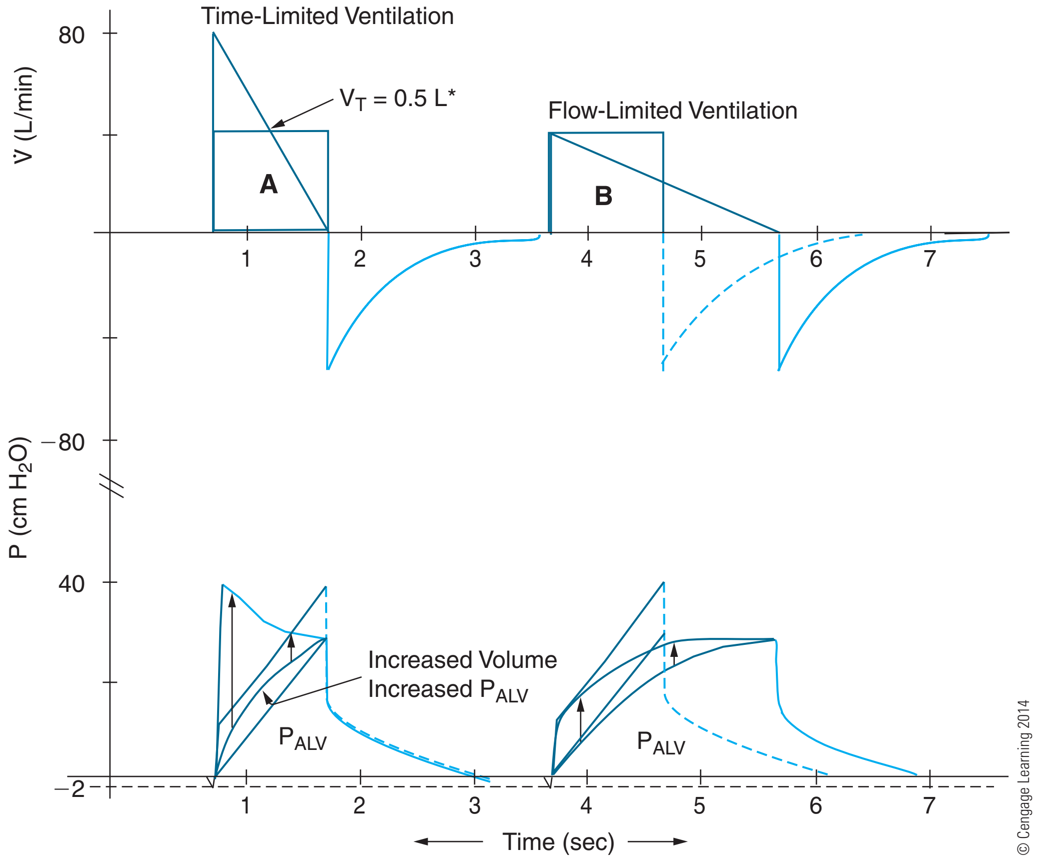

FIGURE 11-14 Changes of dynamic pressure and alveolar pressure (P_{ALV}) waveforms created by descending ramp flow during time-limited (left) and flow-limited (right) ventilation. *(See Table 11-7 for explanations of 0.5 L in the first half of inspiration during true DRFW)

of inspiration, because flow-resistive pressure (P_{TA}) is relatively constant and always "stacked" on top of the peak P_{ALV} by end-inspiration, In the second example (top right), the *initial* peak flow stays the same for both constant-flow and descending-flow ventilation during flow-limited ventilation.

Figure 11-14 delineates the DRFWs (solid lines) into their component parts of P_{TA} (arrows) and P_{ALV} superimposed for comparison over the P_{TA} and P_{ALV} areas for CFWs (dashed lines). The P_{TA} and P_{ALV} for the CFWs rise linearly. Like constant-flow ventilation, the rise in P_{ALV} during descending ramp flow ventilation is dependent on volume delivered and C_{LT}. Lung pressure rises exponentially for descending ramp flow ventilation, and pressure rise is greater in the first half of inspiration because greater volume accumulates in the lung compared to constant-flow ventilation, but flow-resistive pressure steadily subsides in correspondence with reduction in flow. By observation the volume delivery (area under the flow wave) for the DRFWs decreases per unit time. Calculation of the volume under a true DRFW shows that 75% of the volume is delivered during the first half of inspiration. The flow waveforms in Figure 11-14 are the same as in Figure 11-13.

✔ (Figure 11-14) In constant-flow and descending ramp flow ventilation, the rise in alveolar pressure (P_{ALV}) is directly related to the volume delivered and inversely related to the compliance.

As shown in Tables 11-5 and 11-6, the V_T for the CFW (40 L/min or 0.67 L/sec) is 0.67 L, and therefore, 0.33 L is delivered in the first half of inspiration. Table 11-7 shows that the volume delivered in the first half of inspiration for the DRFW is 0.5 L.

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TABLE 11-7 Volume Delivered in the First Half of Inspiration for the True DRFW Compared to the CFW (see Figure 11-14)

True DRFW Volume in the first half of inspiration:	% volume in the first half of inspiration:
Volume = Average Flow × T_I	
Volume = ½ (PF L/sec + *EF L/sec) × ½ T_I (sec)	80 L/min × min/60 sec = 1.33 L/sec
Volume = ½ (1.33 L/sec + 0.67 L/sec) × ½ (1.0 sec)	40 L/min × min/60 sec = 0.67 L/sec
Volume = ½ (2 L/sec) × 0.5 sec	V_T = 0.67 L for DRFW
Volume = 0.5 L	0.5 L/0.67 L × 100% = 75%
CFW Volume in the first half of inspiration:	% volume in the first half of inspiration:
Volume = Average Flow × ½ T_I	V_T = 0.67 L for CFW
Volume = 0.67 L/sec × ½ (1.0 sec)	0.33 L/0.67 L × 100 = 50%
Volume = 0.67 L/sec × 0.5 sec	
Volume = 0.33 L	

**End-flow for the DRFW in the first half of inspiration = 40 L/min or 0.67 L/sec*

Given that T_I is held constant in the first example of descending ramp flow compared to constant flow ventilation, greater volume (75% vs. 50%) is delivered by the DRFW, and therefore, there is a faster rise in P_{ALV} in the first half of inspiration in the first pressure waveform for the DRFW compared to the rise in P_{ALV} for the CFW. In the second half of inspiration, the slope of the rise in P_{ALV} for the DRFW is lower than for the CFW because only 25% of the V_T is delivered for the DRFW compared to 50% of the V_T delivered for the CFW. There may be an improvement in oxygenation using the DRFW compared to the CFW, according to research, because the alveoli are more distended throughout inspiration. Given the same T_I and V_T, mean P_{ALV} is higher during descending ramp flow compared to constant flow ventilation.

✓ Given the same T_I and V_T, mean P_{ALV} is higher during descending ramp flow compared to constant flow ventilation.

The same concepts explored above for the DRFW can be applied to the P_{ALV} pattern presented in the second pressure waveform in Figure 11-14, but the comparison of pressures for the waveforms is a little more complicated. As stated, the ventilator holds peak flow constant for the adjustment in flow waveforms. In this second example, however, the P_{ALV} slope for the CFW is higher throughout inspiration compared to the P_{ALV} slope of the DRFW's pressure waveform. Since T_I is extended for descending ramp flow ventilation to take place, the entire V_T is delivered by the CFW within the time period for which only 75% of the V_T is delivered by the DRFW. Thus, given this condition, pressure-rise is steeper for the CFW. But also note that in the second example, as in the first, because there is a proportionate decrease in volume delivered per unit time during descending ramp flow ventilation, the P_{ALV} generated per unit time steadily decays during inspiration compared to the linear rise in pressure over time for the CFW. Thus, Figure 11-14

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demonstrates that the slope for the P_{ALV} curve generated during descending ramp flow ventilation proportionately decreases per unit time as the summative P_{ALV} increases. The result is that a convex (progressive decay) pressure pattern is always created during inspiration for descending ramp flow ventilation, which is consistent with the pattern of volume delivery.

Peak Flow and Tidal Volume Relationship in Time-Limited Ventilation

Figure 11-15 demonstrates descending ramp flow ventilation where T_I is maintained but flow is reduced. The first example (on left) shows that the P_{TA} is high for the set flow rate (80 L/min). Given the same lung and circuit characteristics for each set of waveforms, the second example (on right) shows the results to the pressure pattern if T_I is maintained (time-limited ventilation), but flow is reduced. Given the same T_I and a reduction in flow, the V_T is reduced (V_T = Average Flow × T_I). The consequence of the reduction in flow is that V_T is reduced, as is the P_{TA} and P_{ALV}, causing a change in the pattern. With the reduction in the initial P_{TA}, the pressure pattern is relatively constant and PIP is no longer at the beginning of inspiration.

✓ (Figure 11-15) During time-limited ventilation (constant T_I), a decrease in flow causes a lower V_T, P_{TA}, and P_{ALV}.

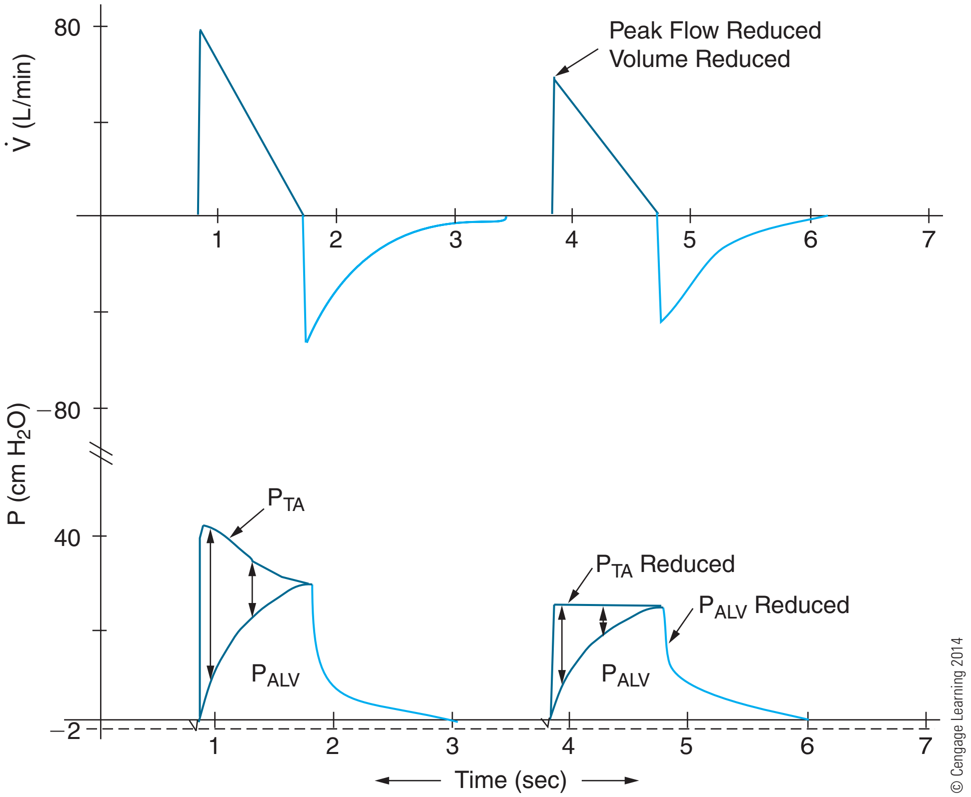

FIGURE 11-15 Characteristic changes of P_{TA} and P_{ALV} waveforms created by changes in peak descending ramp flow during time-limited ventilation. Decrease of peak descending ramp flow reduces the volume delivered, P_{TA}, and P_{ALV}.

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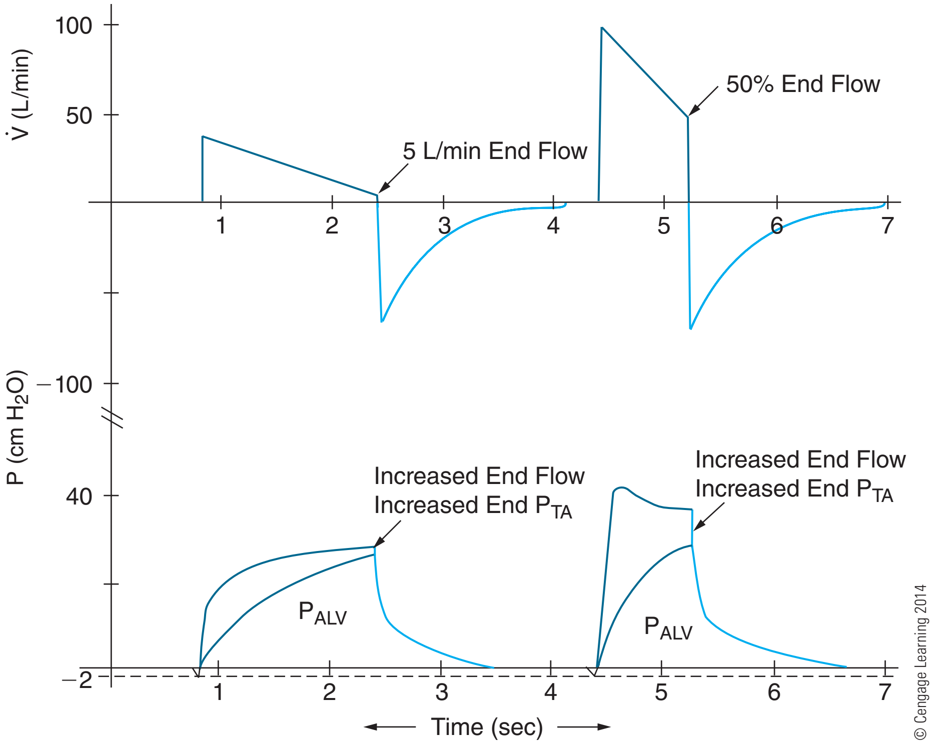

FIGURE 11-16 Effects of end flow on the pressure waveforms during descending ramp flow ventilation. Figure shows two end flows: 5 L/min (left) and 50% of peak inspiratory flow (right). Increase of end flow raises the transairway pressure (P_{TA}), but not the alveolar pressure (P_{ALV}).

Effects of End-Flow on End-Transairway Pressure

✔ (Figure 11-16) During descending ramp flow ventilation, a higher end-flow raises the P_{TA} at end-inspiratory, but not the alveolar pressure (P_{ALV}).

Figure 11-16 demonstrates the changes in P_{TA} patterns that could be expected when end-flows for DRFWs are increased. In the first example, the end-flow is 5 L/min (PB840). The pressure pattern shows a slight increase in the P_{TA} above the P_{ALV} at the end of inspiration as a result of the slight resistance to end-flow being above baseline. The second set of waveforms shows a 50% DRFW where end-flow is 50% of the initial peak flow set. The initial flow set is very high (100 L/min), which causes an exponential rise in initial P_{TA} (40 cm H_2O) because of turbulent flow and circuit/airway resistance. The end-P_{TA} is also relatively high from resistance to flow above the peak P_{ALV} compared to the first example since end-flow is 50 L/min (50% of 100 L/min) compared to 5 L/min. These flow and pressure patterns do not represent ventilation of the same patient. Peak P_{ALV} is at the same level, but V_T delivered is much greater for the second flow waveform example compared to the first.

Distribution of Delivered Tidal Volume

In Figure 11-17, volume-time waveforms have been added for comparison with DRFW and pressure-time waveforms. Since flow rate is highest in the beginning of inspiration for a DRFW, the slope in rise to peak volume, like P_{ALV}, is initially

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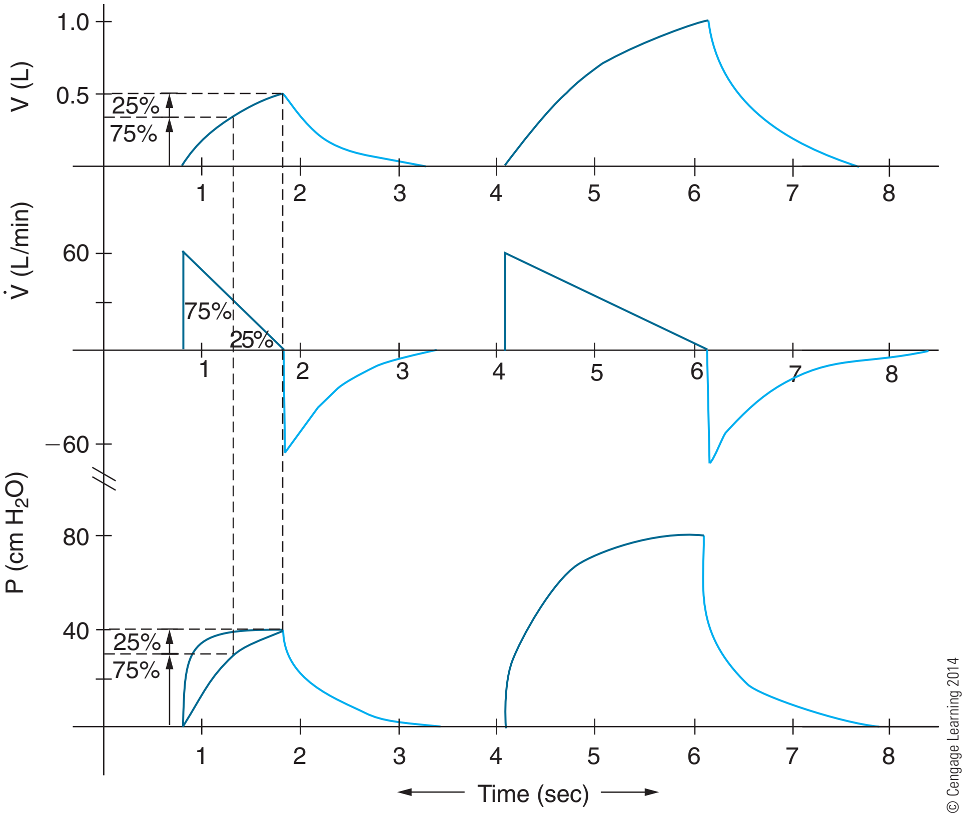

FIGURE 11-17 Effects of inspiratory time using descending ramp flow on the delivered tidal volume (V_T) and peak inspiratory pressure (PIP). An increase of inspiratory time from 1 (left) to 2 sec (right) raises the V_T and PIP.

fast and becomes proportionately reduced over time as flow descends. In the first example, peak flow is set at 60 L/min and T_I is 1 second, and the volume delivered is 0.5 liter [1/2 (peak flow × I time)]. The volume delivered in the first half of inspiration is 0.375 L (75% of 0.5 L). The volume delivered in the second half of inspiration is 0.125 L (25% of the remaining V_T). See Table 11-7 for calculation of volume delivered in the first half of inspiration during true DRFW.

The pressure-time pattern in the first example in Figure 11-17 shows the development of P_{ALV} in correspondence with delivery of the V_T. Determining the P_{ALV} curve requires knowing the volume delivered per time and the total C_{LT}. Because there is no flow at end-inspiration, there is no P_{TA} at end-inspiration, and the PIP represents the peak P_{ALV}, which is 40 cm H_2O. Total C_{LT} is 0.0125 L/cm H_2O or 12.5 mL/cm H_2O (C_{LT} = volume/P_{ALV} = 0.5 L/40 cm H_2O). If there is no change in C_{LT} during V_T delivery, P_{ALV} halfway through inspiration will depend on the volume delivered by that time. The example shows, as discussed earlier, that 75% of the V_T is delivered in the first half of inspiration; consequently, 75% of the peak P_{ALV} will be attained, which is 30 cm H_2O (75% × 40 cm H_2O), or P_{ALV} is equal to volume calculated for that time divided by total C_{LT} (P_{ALV} = 0.375 L/0.0125 L/cm H_2O = 30 cm H_2O). The remaining 25%

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of the P_{ALV} is developed in the second half of inspiration ($P_{ALV} = 25\% \times 40$ cm $H_2O = 10$ cm H_2O, or $P_{ALV} = 0.125$ L/0.0125 L/cm $H_2O = 10$ cm H_2O). Thus, any point along the P_{ALV} curve can be predicted and plotted by calculating the volume delivered per time provided that total C_{LT} is known. This calculation for DRFW also applies to CFW.

In the second example (Figure 11-17), peak flow is maintained at 60 L/min, but V_T is doubled [1/2(60 L/min]/60s/min × 2s = 1 L], which increases T_I to 2 sec. As a result of doubling V_T, peak P_{ALV} is doubled ($P_{ALV} = 1.0$ L/0/0125 L/cm $H_2O = 80$ cm H_2O). Note that the initial flow-resistive pressure is sustained in the second pressure waveform since the initial flow has not changed.

CMV during Descending Ramp Flow Ventilation

✔ The end-flow during descending ramp flow ventilation is zero. The alveolar pressure (P_{ALV}) or plateau pressure at this point equals the end-inspiratory pressure. End-inspiratory pause is not needed to obtain the P_{ALV}.

Figure 11-18 is an example of CMV during descending ramp flow ventilation. The patient initiates the first two breaths and the ventilator triggers the next two breaths. During descending ramp flow ventilation, the peak alveolar pressure (peak P_{ALV}, or plateau pressure) and C_{LT} can be calculated without the need for an end-inspiratory pause. This is because at end-inspiration, the flow is zero (i.e., similar to end-inspiratory pause) and the peak alveolar pressure remains in a plateau state.

Figure 11-18 shows descending ramp flow ventilation with a peak flow of 60 L/min or 1 L/sec. For an inspiratory time of 0.5 sec, the tidal volume is 500 mL (1 L/sec × 0.5 sec). With an end-flow pressure of 35 cm H_2O, the calculated C_{LT} is 14.3 mL/cm H_2O (500 mL/35 cm H_2O).

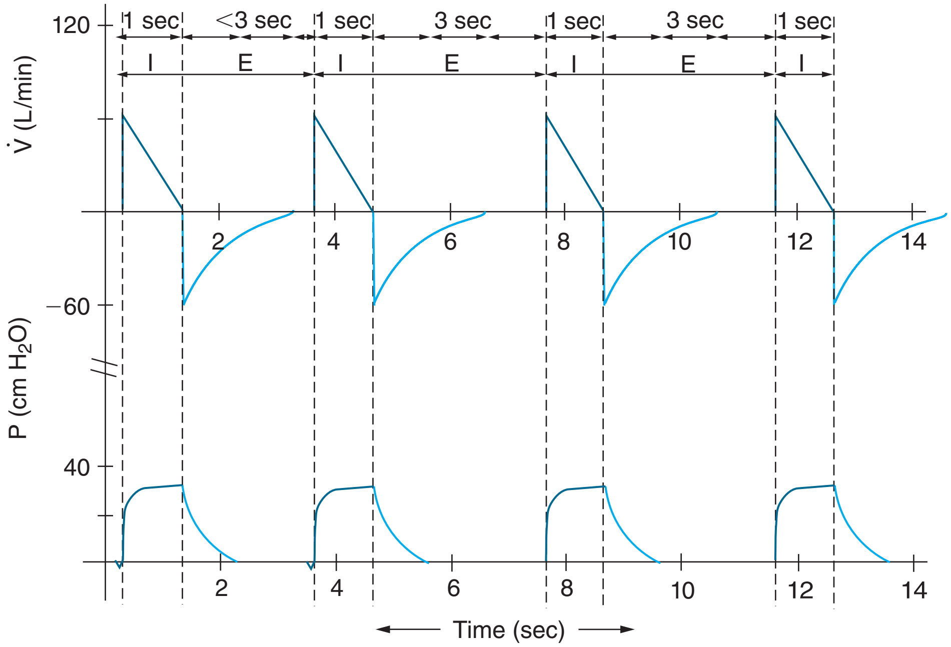

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FIGURE 11-18 Assist (first and second waveforms) and mandatory (third and fourth waveforms) mechanical ventilation during descending ramp flow ventilation.

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WAVEFORMS DEVELOPED DURING PRESSURE-CONTROLLED VENTILATION

Pressure-Controlled Ventilation (PCV)

Figure 11-19 is an example of pressure-controlled ventilation (PCV) in the CMV mode. In PCV CMV, a set (operator) pressure is targeted and sustained by the ventilator for a set T_I period. Also, the frequency, T_I or I:E ratio are set by the operator. The V_T and flow rate delivered by the ventilator are variable, and are dependent on the pressure level set and the patient's lung characteristics. The ventilator may be time- or patient-triggered and delivers a DRFW in a manner that will reach and sustain the pressure level set. In Figure 11-19, the pressure level set for ventilation is 30 cm H_2O. For the letters on the pressure-time waveform, *a* marks the end of expiration and beginning of inspiration; *b* indicates the PIP level set and sustained; *c* is the transition from inspiration to expiration; and *d* is end of expiration with flow. **Total cycle time (TCT)** is 2 seconds and frequency is 30/min (60s/2s/breath = 30/min). T_E is twice T_I. The I:E ratio set is 1:2.

total cycle time (TCT): Time period from beginning inspiration to the beginning of next inspiration.

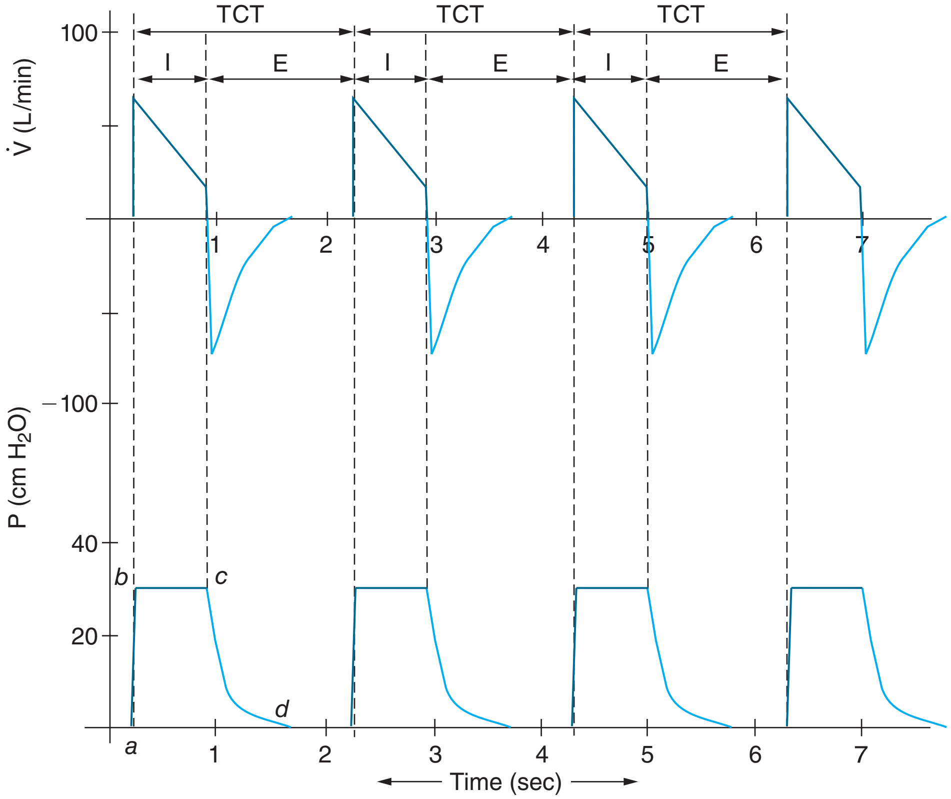

FIGURE 11-19 Characteristics of pressure-controlled ventilation (PCV). The pressure control level of 30 cm H_2O is set and sustained at this level throughout the inspiratory phrase. The flow patterns may vary according to the compliance and airflow characteristics.

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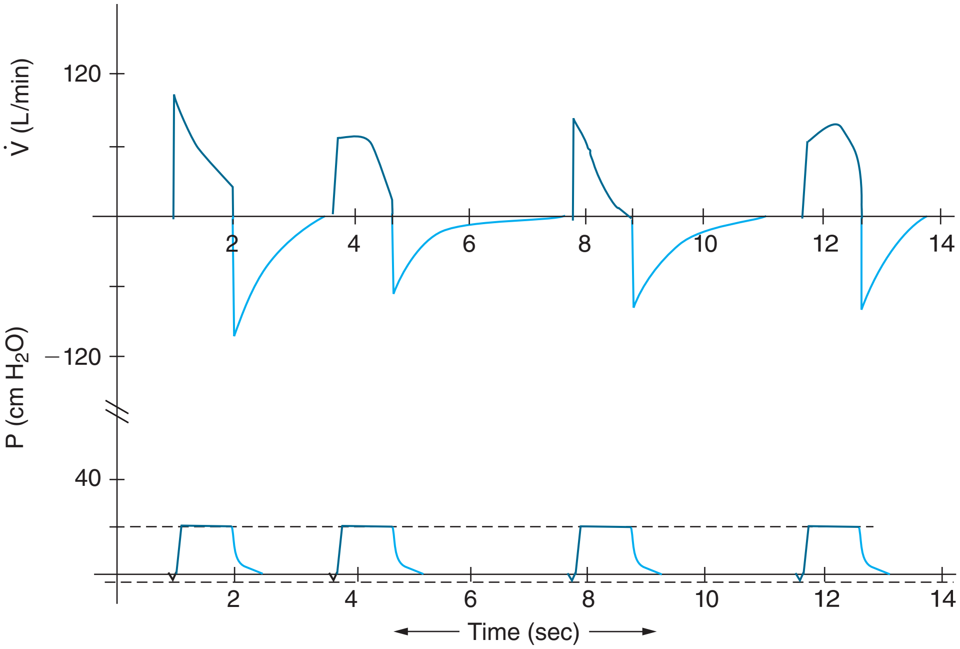

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FIGURE 11-20 Effects of changing compliance and airflow resistance on the flow and delivered volume during PCV. The pressure waveforms remain unchanged during PCV. Flow waveforms change in response to the fluctuating compliance and airflow resistance characteristics. Since the area under each flow waveform reflects the delivered volume, the volume changes accordingly.

Assist Breaths during Pressure-Controlled Ventilation

✓ During PCV, the pressure level, rate, and I:E ratio are preset. Flow and V_T are dependent on the pressure level, patient effort, and lung/airway characteristics.

✓ During PCV, the flow wave patterns may fluctuate based on the patient's respiratory requirement. The V_T is dependent on the pressure level, patient effort, and lung/airway characteristics.

Figure 11-20 is a theoretical example of a patient who is air-hungry, not relaxed, has variable flow demands, and is triggering all the breaths during PCV. By definition, PCV should sustain the pressure level set regardless of variable flow demand and lung characteristics. In this example the flow pattern fluctuates with each breath, so the V_T would also be fluctuating. The PIP is set at 20 cm H_2O, and is sustained (dashed line) by the ventilator. For the pressure level to be sustained, the ventilator would have to vary the flow to maintain the pressure set. Historically, pressure control has always offered assisted breaths, but it has not offered such flexibility in flow delivery, and such variable flow demands would likely cause dyssynchrony and necessitate an increase in the pressure level set to meet the patient's flow and volume demands. Pressure support ventilation, discussed later, demonstrates a greater capacity for accommodating variable flow demands while maintaining the pressure set on the ventilator.

Inverse Ratio Pressure-Controlled Ventilation (IRPCV)

inverse ratio pressure-controlled ventilation (IRPCV): A mode of ventilation in which the peak inspiratory pressure is preset and the I time is longer than the E time.

Figure 11-21 demonstrates **inverse ratio pressure-controlled ventilation (IRPCV)** set at 20 cm H_2O with 10 cm H_2O of PEEP for a combined pressure (PIP)

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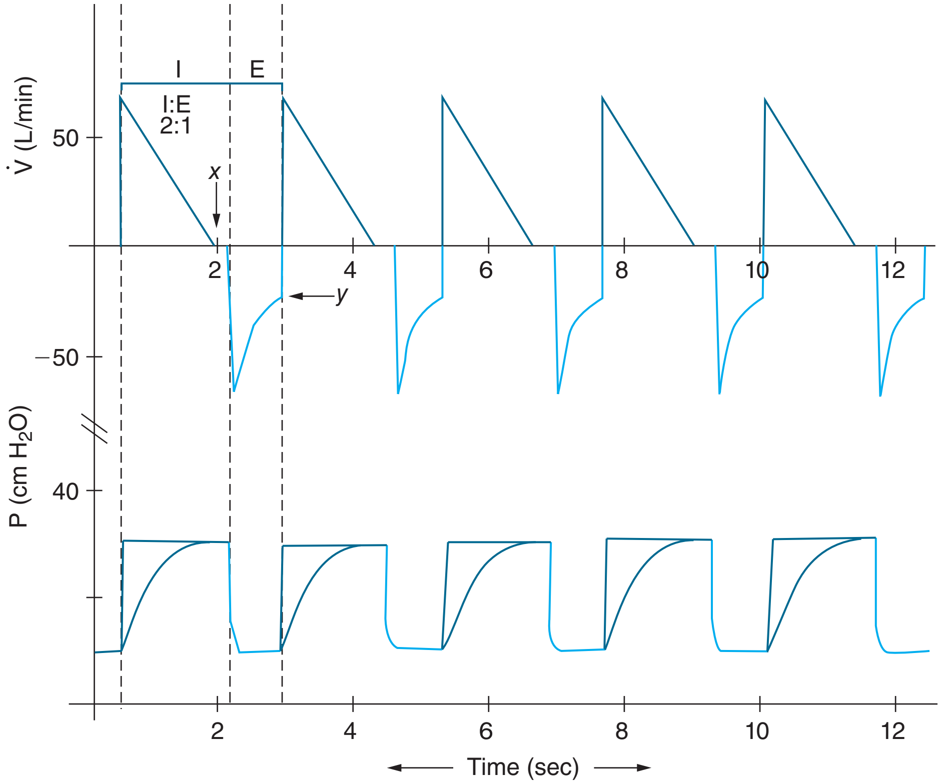

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FIGURE 11-21 Inverse ratio pressure-controlled ventilation (IRPCV). Arrow *x* shows end of inspiratory flow because the pressure control level has been met. Arrow *y* shows incomplete exhalation, air trapping, and the potential for development of auto-PEEP.

of 30 cm H_2O. IRPCV is used under conditions of severe hypoxemia and lung injury (ARDS). Since an inverse ratio respiratory pattern is abnormal and uncomfortable, patients are sedated and paralyzed to prevent them from "fighting" the ventilator. In this example, inspiratory flow drops to zero (arrow *x*) since the pressure target has been met. The ventilator is maintaining the set pressure level by closing the expiratory valve and curtailing flow. This process holds gas in the patient's lungs until end-inspiratory time is reached. The PIP during this no-flow time period is, in effect, a pause pressure and peak P_{ALV}. The dashed lines applied to the pressure waveforms indicate the development of the P_{ALV} during inspiration and shows that peak P_{ALV} is reached when flow ends. The volume held in the patient's lungs depends on the patient's C_{LT}. If the expired V_T is monitored, the patient's lung compliance can be measured [$C_{LT} = V_T/(PIP - \text{Total PEEP})$]. Total PEEP includes PEEP set plus intrinsic or auto-PEEP. Arrow *y* at the first expiratory flow wave shows that auto-PEEP is being created because the ventilator time triggers successive breaths before the exhalation is complete. Since the expiratory flow is unable to descend to the baseline, gas is trapped in the lungs. This causes elevation of the functional residual capacity and reduction of V_T delivered. During PCV, the level of auto-PEEP proportionately reduces the V_T delivered to the lungs. Ventilation is compromised, which may cause or worsen hypercapnia.

✓ IRPCV offers an unnatural breathing pattern and may be uncomfortable. The patient is often sedated and paralyzed to prevent patient-ventilator dyssynchrony.

✓ IRPCV may be helpful in conditions of severe hypoxia and lung injury (e.g., ARDS).

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✔ Graphics can be used to titrate and increase the T_I to an appropriate inverse-ratio level without causing gas trapping or auto-PEEP.

Based on research, auto-PEEP should be avoided if possible, and external PEEP applied instead. A valuable advantage of having graphics is that auto-PEEP can be readily observed and prevented whenever lung conditions have changed or ventilator settings have been altered. Graphics can be used to titrate and increase the T_I to an appropriate inverse-ratio level without causing gas trapping or auto-PEEP. This management technique serves to increase mean P_{ALV} and improve oxygenation at lower peak P_{ALV} to reduce the potential of barotraumas.

PRESSURE SUPPORT AND SPONTANEOUS VENTILATION

Pressure Support Ventilation (PSV)

✔ In PSV, only the pressure support level is preset. The V_T depends on the PS level, inspiratory effort, and patient's lung/airway characteristics.

Figure 11-22 shows different features of pressure support ventilation (PSV). The first set of waveforms shows the patterns that develop for a relaxed patient breathing synchronously with the ventilator, a concave flow- and square pressure-time

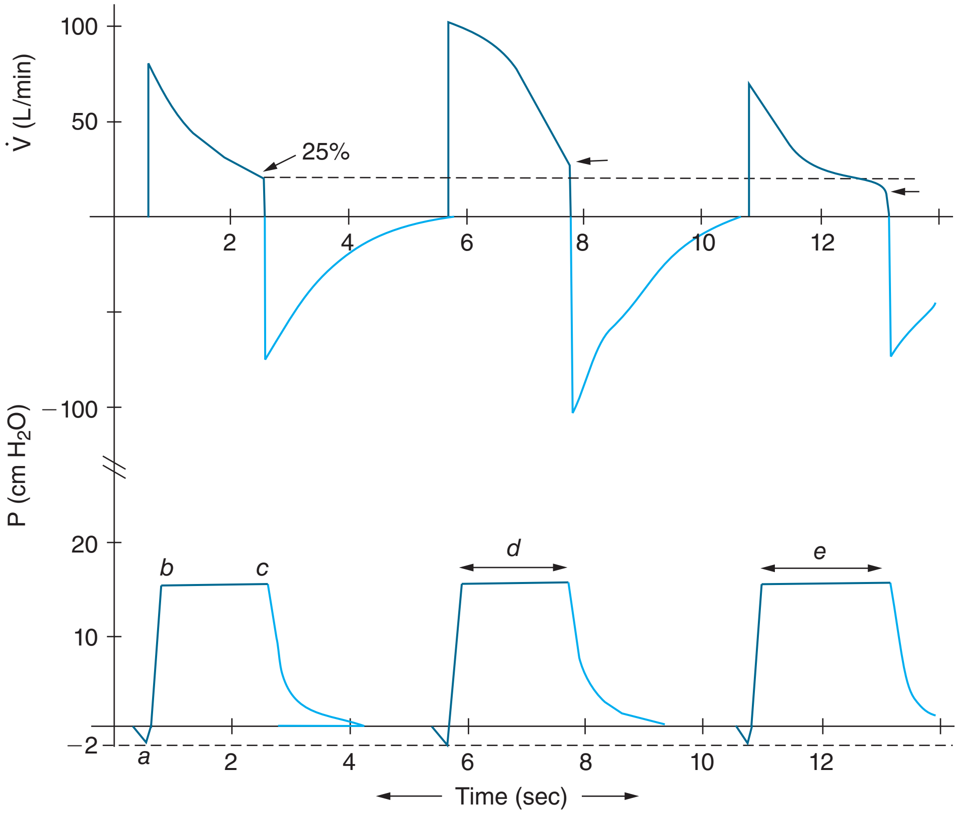

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FIGURE 11-22 Characteristics of pressure support ventilation (PSV). The pressure support level remains constant as long as the inspiratory flow continues and is above the end flow. The inspiratory flow and inspiratory time are dependent on the patient's spontaneous breathing effort. The arrows show an end flow of 25% of the peak inspiratory flow. When the inspiratory flow reaches 25% of the peak inspiratory flow, pressure support (inspiration) ends and expiration begins.

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✓ During PSV, the flow, volume, and inspiratory time are under the patient's control.

pattern. PSV is pressure-controlled ventilation (Chatburn 2001, 2007) in the spontaneous mode of ventilation. Although each breath is mechanically supported by a set pressure, each breath requires a "spontaneous effort" by the patient throughout most of inspiration to sustain flow delivery above cycling (termination) level. Thus, flow, volume, and inspiratory time are primarily under the patient's control, which defines the mode of ventilation as *spontaneous*. However, the pressure can be set high enough to limit virtually all effort by the patient except for triggering the breath.

If the patient inspires in a relaxed manner as presented in the first set of waveforms, gas reaches a peak flow level causing the pressure to reach the preset pressure support level. The peak flow then tapers to sustain the preset pressure. However, T_I is not constant because the flow will continue until it reaches a ventilator-specific preset level (e.g., 5% or 25% of initial peak flow) to terminate the flow (and breath).

For the first pressure waveform example, letter *a* indicates it is an assist, pressure-/patient-triggered breath; *b* indicates that the pressure level set, 15 cm H_2O, has been quickly reached; and *c* shows that the pressure target has been sustained throughout inspiration. The flow wave demonstrates that the ventilator provides flow at a high level initially to exceed the patient's flow demands, in order to reach the set pressure level. The end-flow per breath (dashed line) shows that each wave is flow-cycled into expiration at 25% (arrows) of the initial peak flow rate. The peak and end-flow may vary with each breath.

The second flow wave demonstrates that a higher flow is provided to meet the patient's increased demand, but the set pressure level is maintained. The third flow wave shows a lower patient demand and the flow is sustained until 25% of the initial flow is reached. In comparison of all three waveforms in Figure 11-22, the inspiratory flow and inspiratory time can be variable (e.g., *d* and *e* in figure), depending on the patient's spontaneous breathing effort and lung/airway characteristics.

Adjusting Rise Time during PSV

✓ In PSV, the faster the initial rise to peak flow (short rise time), the sooner the preset PS level is reached. This may cause the ventilator to overshoot the PS limit.

✓ A slow rise time during PSV is more comfortable for the patient.

Figure 11-23 demonstrates PSV at 10 cm H_2O with PEEP of 5 cm H_2O. The first set of flow and pressure waveforms is ideal. The second flow waveform demonstrates a rapid rise in initial flow that causes a spike in the initial pressure that overshoots the set pressure limit. The pressure spike at the beginning of inspiration (arrow *x*) can cause patient discomfort and increased work of breathing (Dick et al., 1996).

Letters *a* and *b* flow waves demonstrate a feature called rise time percent (%), which is the percentage of the inspiratory cycle time required to reach the peak flow rate and the pressure target set. The vertical dashed lines preceding the flow waveforms show that the rise to peak flow takes a progressively longer period of time. The dashed lines on the third and fourth pressure waveforms were added to show that the slope or rise to the set pressure is progressively slower as a result. The faster the rise in initial flow, the sooner the set pressure is reached. An advantage of a slower rise to the peak flow rate and set pressure levels is that greater comfort is provided.

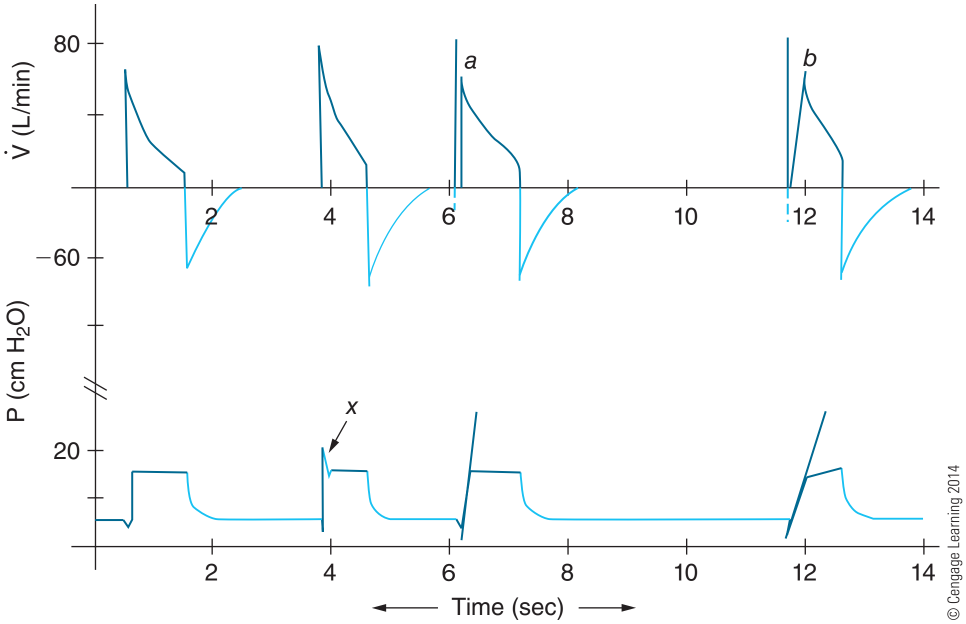

FIGURE 11-23 PSV at 10 cm H_2O of pressure support over 5 cm H_2O of PEEP. Arrow *x* shows a rapid rise in initial inspiratory flow generated by the patient causes a spike in the initial pressure that overshoots the pressure support level. Letters *a* and *b* show two different rise time percents. A slower rise time percent (*b*) causes a more gradual rise to the pressure support level and it provides more patient comfort.

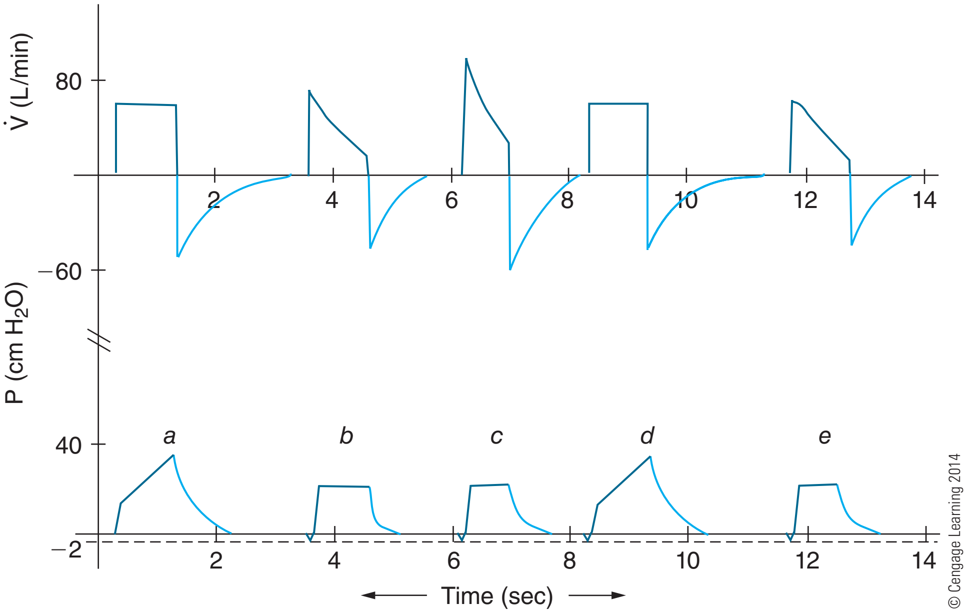

FIGURE 11-24 SIMV and PSV during constant flow ventilation. Mandatory breath (*a*); pressure support breaths (*b*, *c*, and *e*); assist breath (*d*).

SIMV (CFW) and PSV

Figure 11-24 exemplifies the combination mode, SIMV + PSV. Two ideal CFWs and corresponding step ascending ramp pressure patterns, *a* and *d*, depict volume-controlled

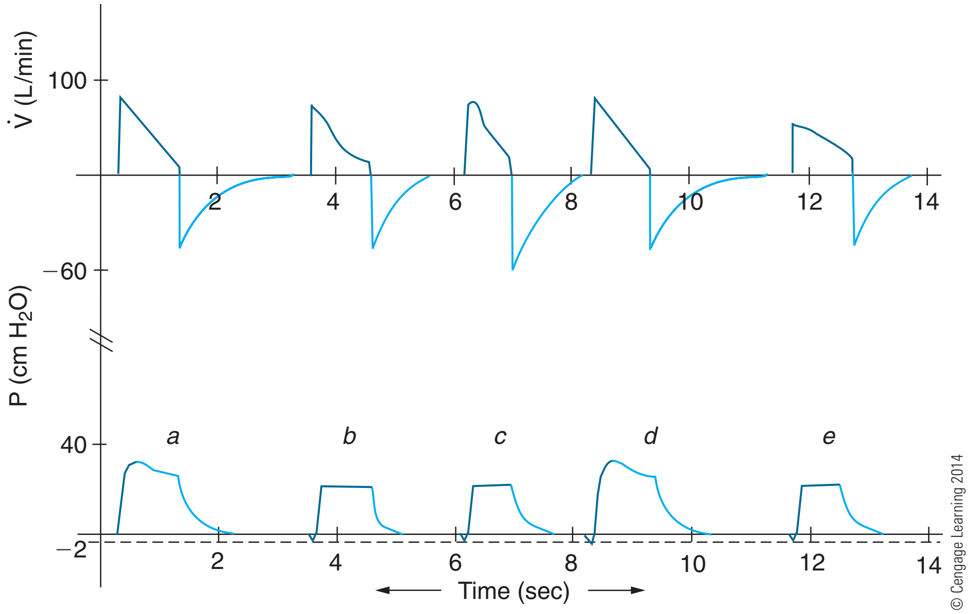

FIGURE 11-25 SIMV and PSV during descending ramp flow ventilation. Mandatory breath (*a*); pressure support breaths (*b*, *c*, and *e*); assist breath (*d*).

breaths, the first mandatory and the other an assist breath. Three pressure-supported breaths, *b*, *c*, and *e*, show variable DRFWs, patient-triggered, pressure-controlled (20 cm H_2O) breaths of varied flow, volume, and inspiratory time.

SIMV (DRFW) and PSV

Figure 11-25 depicts the combination mode, SIMV + PSV, except that DRFWs with end-flows at 5 L/min have replaced the CFW used in Figure 11-24. The DRFWs for the SIMV breaths show that initial flow was set relatively high (80 L/min), resulting in PIP for pressure patterns *a* and *d* being created at the beginning of inspiration. Three pressure-supported breaths, *b, c*, and *e*, are displayed again for this example.

EFFECTS OF LUNG CHARACTERISTICS ON PRESSURE-CONTROLLED VENTILATION WAVEFORMS

✔ During pressure-controlled ventilation, the pressure target is sustained while the flow and volume vary when there are changes in pulmonary mechanics.

Figure 11-26 exemplifies the effect that changes in circuit/airway resistance and C_{LT} can have on waveforms, flow, and volume delivered during pressure-controlled ventilation. During CMV VCV, volume delivery is sustained and the pressures (P_{TA}, P_{ALV}, or both) vary when there are changes in pulmonary mechanics. During pressure-controlled ventilation, in either the CMV or spontaneous modes, the pressure target is sustained while the flow and volume vary when there are changes in pulmonary mechanics. For each pressure-controlled breath under the dashed lines labeled *A* and *B* (Figure 11-26) characterize different lung conditions during PCV. The peak flow and area under the inspiratory curve of the flow waveforms

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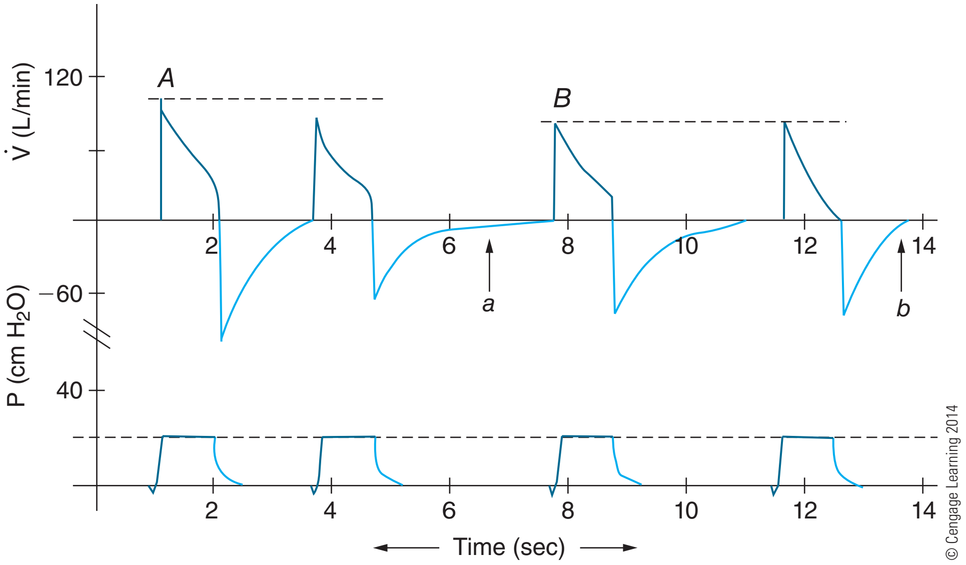

FIGURE 11-26 During pressure-limited ventilation, the flow and volume are dependent on the airflow and compliance characteristics of the patient/ventilator system. A higher flow and larger volume are observed in *A* and *B* (first and third breaths) than their adjacent breaths (second and fourth breaths). The waveforms show that *A* has a lower airflow resistance and *B* has a higher compliance than their adjacent breaths.

(first and third) are comparatively greater than the adjacent flow waveforms (second and fourth) in examples A and B. Thus, greater volume is being delivered by the first and third waveforms.

For each pressure-controlled breath under the dashed lines labeled *A* and *B* (Figure 11-26) characterize different lung conditions during PCV. The peak flow and area under the inspiratory curve of the flow waveforms (first and third) are comparatively greater than the adjacent flow waveforms (second and fourth) in examples *A* and *B*. Thus, greater volume is being delivered by the first and third waveforms.

✓ During pressure-controlled ventilation, an increased airflow resistance will reduce the delivered flow and tidal volume.

In example *A* (first and second waveforms), given no change in C_{LT} or effort, greater airway/circuit resistance will tend to cause some reduction in the initial or average flow rate, and therefore, a reduction in V_T as demonstrated by the adjacent flow waveform. Greater flow-resistive pressure (P_{TA}) causes the set (target) pressure to be reached at a lower flow rate. A reduction in delivered V_T may be the result unless the average flow is sustained, which may be demonstrated by a reduction in initial peak flow, but an elevation in the end-flow rate. An increase in airway or circuit resistance will result in a reduced V_T because the peak and average flow will be reduced, as demonstrated in this example. Letter *a* demonstrates that the average expiratory flow rate is reduced, causing longer T_E.

Example *B* (third and fourth waveforms) demonstrates that a reduction in the C_{LT} (i.e., ascites, ARDS) will also result in a reduction in V_T. However, the initial inspiratory flow rate is not affected since resistance to flow is not a factor. The initial driving pressure for flow, the PC level set, has not changed, so the initial flow rate is sustained. However, given lower C_{LT}, more pressure is required for the same volume to be delivered, and since pressure is held constant, the result has to be a reduction

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✔ During pressure-controlled ventilation, a reduction in C_{LT} will reduce the V_T.

in the V_T delivered. Compared to example *A*, less volume is delivered as shown by the area under the second inspiratory flow waveform in example *B*. Peak flow is maintained, but the slope or descent in flow rate from peak level is greater. Thus, the average flow and V_T are reduced (V_T = average Flow × T_I). Letter *b* shows that T_E is reduced since less volume is expired. The peak expiratory flow rate is about the same since the resistance to flow is the same, and the driving pressure for expiratory flow, the PC level set, and end-inspiratory lung pressure, was held relatively constant. During pressure-controlled ventilation, the pressure is held relatively constant while the flow and V_T delivered are altered by changing lung/airway characteristics.

USING WAVEFORMS FOR PATIENT-VENTILATOR SYSTEM ASSESSMENT

✔ Patient-ventilator dyssynchrony increases the work of breathing and prolongs weaning from mechanical ventilation.

Positive pressure in the lungs is not natural. Too much volume under pressure can cause barotraumas or volutrauma. Too little volume or not enough positive end-expiratory pressure (PEEP) to keep alveoli from collapsing (derecruitment) and then reopening (recruitment) during tidal ventilation can cause lung injuries. Too little respiratory muscle use (disuse atrophy) leads to respiratory muscle weakness, and excessive work of breathing (WOB) leads to respiratory muscle weakness or fatigue. Either way, weaning from mechanical ventilation is delayed.

No research to date suggests that patients triggering all the volume- or pressure-controlled breaths in synchrony with the ventilator will develop respiratory muscle weakness from either too little or too much WOB. Conversely, volumes of research suggest that dyssynchrony during ventilator management may impose excessive WOB and prolong mechanical ventilation (Dick et al., 1996). Research also suggests lack of assisted ventilation (patient-triggered breaths) from prolonged paralysis or sedation as a reason for prolonged mechanical ventilation. The best oxygenation and ventilation should be derived at the lowest volume and least lung pressure if patients are breathing in synchrony with the ventilator, which obviates excess WOB and mitigates the potential for excessive lung pressures. Auto-PEEP can be easily observed and corrected using graphics, which reduces the potential for volume trauma. Also, patient-ventilator synchrony can be easily verified and controlled through waveform analysis.

Patient-Ventilator Dyssynchrony

✔ Dyssynchrony during mechanical ventilation increases the WOB, oxygen consumption, minute ventilation, and myocardial work.

Dyssynchrony during mechanical ventilation increases the WOB, oxygen consumption, minute ventilation, and myocardial work. Dyssynchrony can occur with the assisted (patient-triggered) or controlled (time-triggered) breaths. The first pressure waveform in Figure 11-27 depicts a detailed analysis of an assist (patient-triggered) breath. Both pressure-time waveforms depict the step ascending ramp pattern. Thus, a constant (square) flow pattern must have been set to produce these pressure waveforms. The −2 cm H_2O value below baseline (zero) represents the sensitivity threshold level setting. In the first waveform, the patient's effort to trigger the breath

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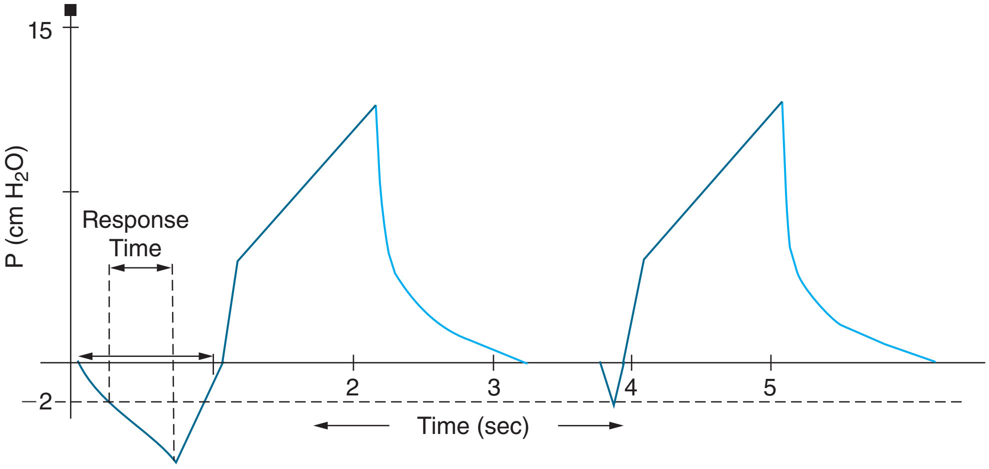

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FIGURE 11-27 Pressure-time waveform demonstrates response time during assist (patient-triggered) ventilation. The patient-triggered negative pressure for the first breath is enlarged to show the response time.

✔ Dyssynchrony can occur with the assisted (patient-triggered) or controlled (time-triggered) breaths.

✔ High ventilatory demand of a patient may be fulfilled with a higher sensitivity setting, a higher peak flow, and use of flow triggering (faster response time for flow delivery).

is exaggerated to analyze the negative pressure deflection in greater detail. The negative pressure deflection crosses the set sensitivity threshold level descending to about −4 cm H_2O before the ventilator is able to respond (deliver gas). How fast the ventilator reacts to a patient's efforts depends on the patient's respiratory drive, sensitivity level set, inspiratory valve response time, and peak flow setting. The response time is measured from the time the sensitivity threshold setting is reached to the time flow is initiated by the ventilator (short, double-headed arrow). Once flow is initiated, pressure changes from a negative to a positive direction. If a patient has a very weak ventilatory drive, a higher sensitivity setting is needed (e.g., −1 instead of −3 cm H_2O). Flow triggering or a higher peak flow setting may also improve patient-ventilator synchrony.

When analyzing the patient-trigger phase of inspiratory effort, it is important to realize that the patient is always experiencing higher negative intrathoracic and intrapulmonary pressures than those recorded at the ventilator (Dick et al., 1996; Messinger et al., 1995). There is a time lag during breathing efforts for the propagation of negative pressure created in the patient's intrapleural space to traverse the airways and respiratory circuit system, and then reach the pressure transducer in the ventilator. Research has shown that a −5 cm H_2O (versus −2 cm H_2O) sensitivity setting can increase WOB to intolerable levels (Marini et al., 1985). Thus, substantial metabolic work to breathe is required during the negative pressure time period (long, double-headed arrow). Consider, for comparison, that for normal spontaneous breathing, approximately −1 cm H_2O P_{ALV} is incurred during inspiration, and gas is flowing into alveoli throughout the inspiratory effort as the alveoli are expanding, which aids the oxygenation and ventilation process instantaneously. There is no gas movement into the alveoli during the negative deflection time period depicted in Figure 11-27. Gas is simply being decompressed and recompressed until positive pressure is recorded. Thus, during pressure triggering, the negative intrathoracic pressure is greater than the ventilator-recorded negative

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pressure (e.g., -10 cm H_2O intrathoracic versus -2 cm H_2O at ventilator). Under this condition, the metabolic work (O_2 consumption) can be greatly increased. Oxygenation and ventilation are being compromised compared to normal spontaneous breathing, especially if sensitivity is set too low.

Dyssynchrony during Constant Flow Ventilation

✔ (Figure 11-28) Insufficient flow (letter *a*) or insufficient tidal volume (letter *b*) may cause patient-ventilator dyssynchrony.

✔ Signs of patient-ventilator dyssynchrony include tachypnea, agitation, use of accessory respiratory muscles, active expiration, paradoxical breathing, and respiratory alternans.

✔ Tachypnea and a general appearance of agitation may be caused by other factors such as pain and psychologic stress.

A lack of ventilator sensitivity to the patient's inspiratory effort may lead to patient-ventilator dyssynchrony as presented in examples *a* and *b* in Figure 11-28. The graphic shows that patient-ventilator dyssynchrony occurs when increased ventilatory demands are not met by a sufficient flow or volume. Whether a particular pattern of dyssynchrony can be proven to be excessive or not is a complicated issue and beyond the scope of this chapter. Perfect waveforms are not necessary. When signs of patient-ventilator dyssynchrony occur, decisions about ventilator management require a competent clinical assessment. The first sign of increased WOB is tachypnea (f $>$20/min) and a general appearance of agitation. Other signs of respiratory muscle stress include use of accessory respiratory muscles, intercostal retractions, and active expiration (use of abdominal muscles). In time, depending on a patient's pulmonary reserve and nutritional status, signs of respiratory muscle weakness or fatigue can develop. Paradoxical breathing pattern is the end result. Unfortunately, the last signs of respiratory muscle weakness or fatigue are blood gas abnormalities showing respiratory failure (Tobin et al., 1986). For this reason, using arterial blood gas results as the primary assessment tool of competent ventilator management is a serious shortcoming.

Tachypnea and a general appearance of agitation may be caused by other factors such as pain and psychologic stress. Patient-ventilator synchrony may not be the problem. But if improvement in patient-ventilator synchrony eliminates the signs of physical and psychologic distress, quality patient care has been enhanced. The appropriate adjustment on the ventilator to improve synchrony is easily facilitated through waveform analysis.

The dashed line for the first pressure-time waveform *a* (Figure 11-28), exemplifies the ideal waveform for a passive patient being mechanically ventilated; that is, the ventilator is generating virtually all the pressure necessary to expand the patient's lungs. The solid line depicts patient-ventilator dyssynchrony and the corresponding waveform created during mechanical ventilation. The greater the drop in pressure and the development of irregular patterns from the ideal pattern (dyssynchrony), the less the ventilator is assisting the patient with breathing. In fact, the dyssynchrony can often impose more WOB onto the patient than that incurred by spontaneous breathing off the ventilator. Physical signs of distress indicate that the patient is enduring too much WOB. If the patient is not on the ventilator because of respiratory failure, some dyssynchrony may not pose a serious problem. Research suggests that it will compromise oxygenation and ventilation compared to optimal, which may be uncomfortable for the patient. Morbidity and mortality outcomes, however, may be the same; the patient is simply doing more of the WOB. But research shows that if patients are recovering from respiratory muscle failure, dyssynchrony

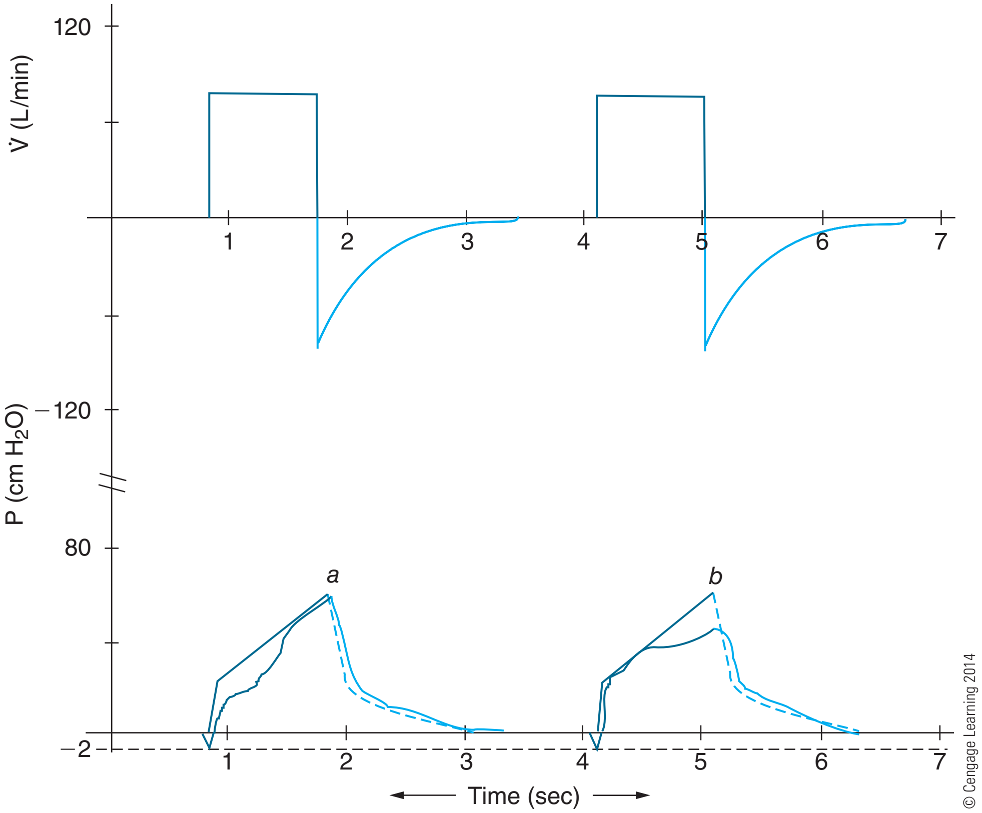

FIGURE 11-28 Constant flow and pressure-time waveforms showing dyssynchronous ventilation as a result of *a*, inadequate initial peak flow, or *b*, inadequate mechanical tidal volume to meet patient demand.

can sustain respiratory muscle weakness or fatigue, and it will prolong mechanical ventilation because recovery time to regain the strength for spontaneous breathing is compromised.

✓ (Figure 11-28) Solid pressure tracing *a* shows insufficient *initial* flow (low peak flow). Solid pressure tracing *b* shows insufficient *end-inspiratory* flow (low tidal volume).

The first pressure-time waveform *a* (Figure 11-28) indicates that a higher initial flow is needed to keep pace with the patient's initial flow demands. The initial flow-resistive pressure is partially removed, so that the pressure to inflate the lungs has been transferred (imposed on) to the patient. The patient is drawing gas (flow demand) from the circuit almost as fast as the incoming gas from the ventilator. As a result, less pressure is created. Increasing the flow rate to a level greater than the patient's demand will provide assistance and restore a normal pressure pattern (dashed curve).

In waveform *b* (Figure 11-28), the initial flow demand appears to be met since the initial rise in pressure is relatively normal. The drop in pressure in this example is at end-inspiration and suggests that the patient needs more flow (i.e., volume) than is being supplied. A drop of pressure at end-inspiration means more volume is being demanded than is being supplied. The ventilator is being time-cycled into expiration before that demand is met.

Sometimes, increasing the peak flow will satisfy the patient's demands in this situation because the volume is delivered sooner. This may cause the patient to relax and breathe in synchrony with the ventilator. Increasing the sensitivity may also

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make it easier to breathe initially, relaxing the patient and satisfying what appeared to be an inadequate peak flow or V_T setting. Experience suggests that all three methods should be attempted. No research shows that there is only one way to satisfy a particular dyssynchronous waveform pattern. These suggestions are based on knowledge, experience, and research. Sometimes only a change in mode from VCV to pressure-controlled ventilation will provide patient-ventilator synchrony. In some patients, the breathing patterns can be so erratic that little can be done to provide synchronous ventilation. Pain, neurologic damage, psychologic stress, or unknown reasons may be causing erratic patterns of breathing that cannot be matched by today's ventilators. Patients may have to be sedated for periods of rest under such circumstances.

✓ Adjusting the peak flow, sensitivity, tidal volume; changing the mode of ventilation; or sedating the patient may restore patient-ventilator synchrony.

Dyssynchrony during Descending Ramp Flow Ventilation

Figure 11-29 presents the same dyssynchronous conditions depicted in Figure 11-28 except that DRFWs are being utilized. The dashed lines show the ideal pressure-time waveforms. Example *a* suggests a higher peak flow setting to meet initially

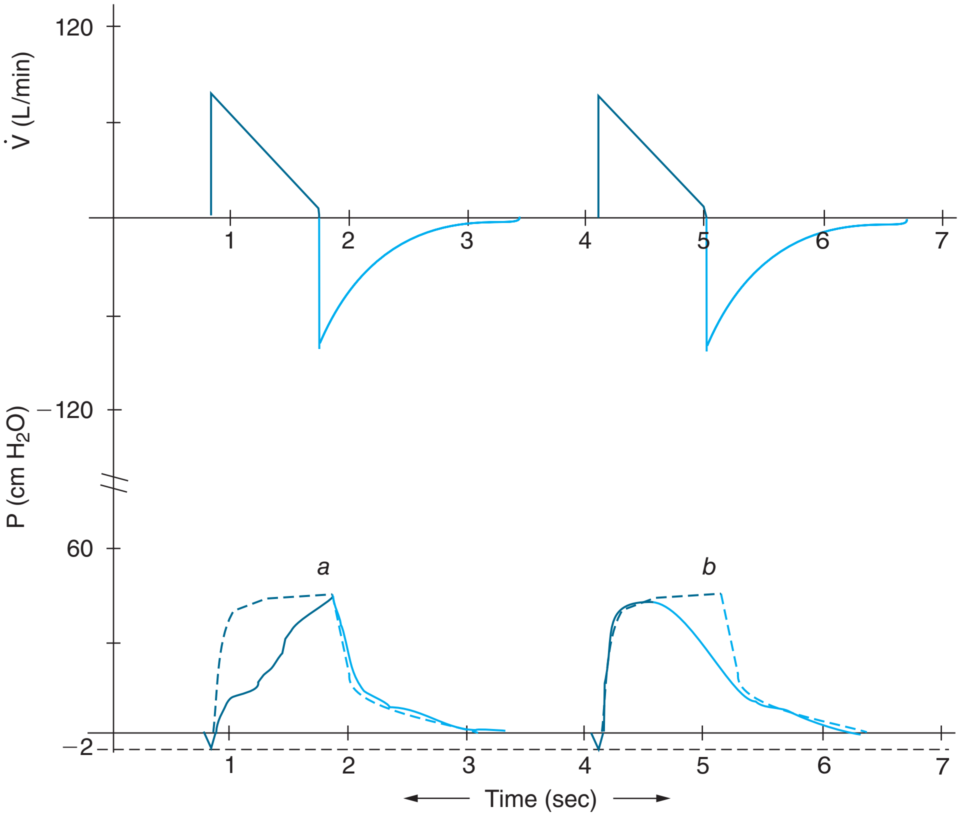

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FIGURE 11-29 Descending ramp flow and pressure-time waveforms showing dyssynchronous ventilation as a result of *a*, inadequate initial peak flow, or *b*, inadequate mechanical tidal volume to meet patient demand.

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high flow demands, and example *b* suggests a need for increasing the V_T. In Figures 11-28 and 11-29, signs of respiratory distress would probably result unless the patient is being sedated.

Changes in Pressure Waveforms during Respiratory Mechanics Measurement

Figure 11-30 shows another important benefit of graphics: assurance that the respiratory mechanics are being accurately measured. These are the same waveforms depicted in Figure 11-4 except that the dashed lines *a* and *b* (arrows) represent possible variations in peak P_{ALV} that could result during a pause in flow. Rather than pressure remaining constant during the pause, pressure either rises above relaxed peak P_{ALV} or descends below it. Pressure rising as in the example (letter *a*) often occurs when a patient tries to expire during the pause time. The procedure (pause at end-inspiration) is unnatural and patients do not always relax, and the neurologic respiratory drive to breathe may prevent it. Dyssynchronous ventilation may be occurring as well, and the patient's breathing pattern does not correspond to end-inspiration in synchrony with the ventilator. There may be many reasons for an

✓ Patient-ventilator dyssynchrony with *rising* airway pressure may occur when the patient moves, talks, coughs, or expires during the pause time.

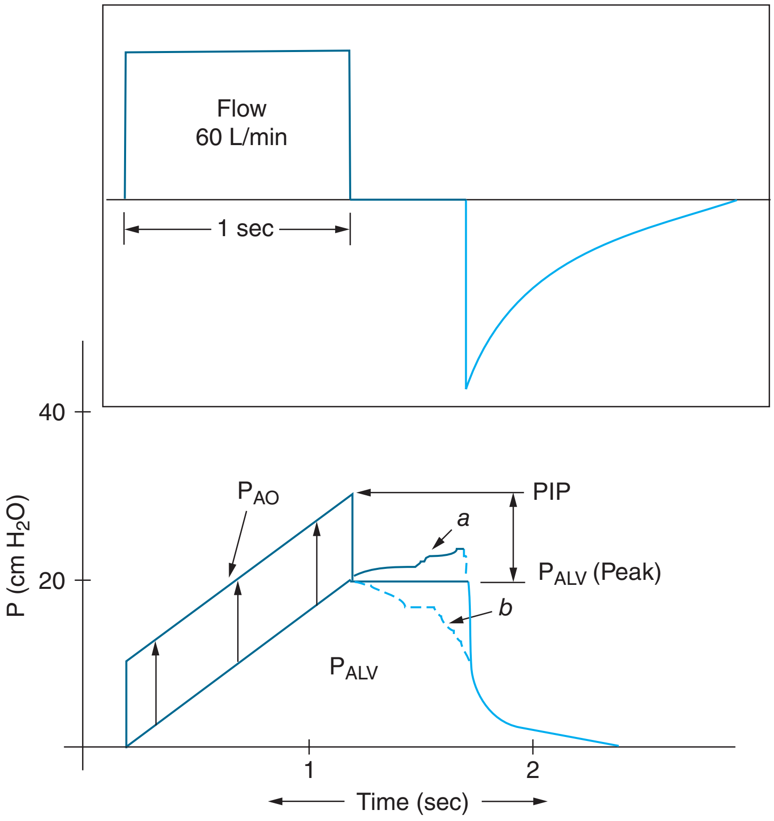

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FIGURE 11-30 Measurements of P_{TA} and P_{ALV} can be affected by the patient's effort to breathe. Dotted line *a* shows patient's expiratory effort during the pause period. Dotted line *b* shows inspiratory effort during the pause period.

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increase in pressure during this period of time. Any movement such as turning or twisting of the thorax can cause pressure to rise. Hands placed on the patient's chest while being attended by a health care provider can increase intrathoracic pressure and peak P_{ALV}. The patient trying to talk or cough will increase P_{ALV}.

Patient-ventilator dyssynchrony with *decreasing* airway pressure may occur with a small leak in the ventilator circuit or when the patient continues to inspire during the pause time.

Letter *b* (Figure 11-30) shows the patient may be trying to continue to inspire, thus expanding thoracic volume, decompressing gas in the system, and dropping pressure. There may be a small leak in the circuit, causing pressure to drop during the pause. Using graphics, you will learn with experience that it is very difficult to obtain accurate respiratory mechanics measurements. Without graphics, errors cannot be observed and may be documented as fact. Patients have to be totally relaxed and passive during the static compliance measurement. Usually, only the CMV mode can be used during respiratory mechanics measurements. Often, the minute ventilation has to be increased 10% to 15% to reduce the patient's $PaCO_2$ to apneic threshold (about 32 mm Hg), to eliminate patient's respiratory drive and induce relaxation, and to obtain valid measurements (Marini et al., 1985, 1986).

Dyssynchrony during Pressure-Controlled Ventilation

Figure 11-31 demonstrates a pressure support level that is set too low to satisfy patient flow or volume demand. The patient's respiratory frequency has increased well above normal (approximately 28/min) with graphic display of dyssynchrony. Physical signs of discomfort and increased work of breathing would undoubtedly

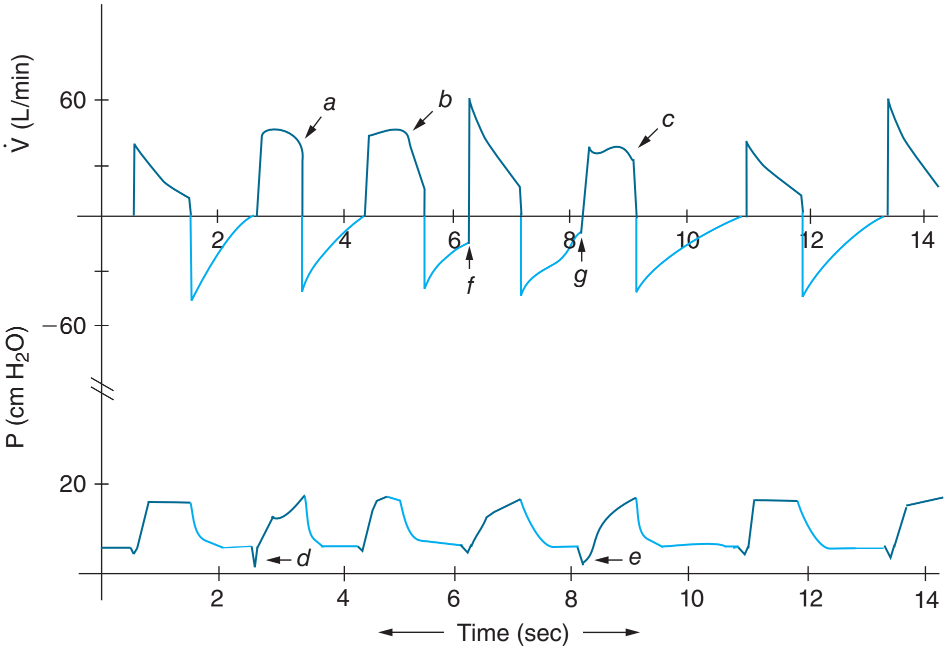

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FIGURE 11-31 An example of pressure support ventilation in normal breaths (first, second, and fifth breaths) and in breaths where patient demands are not being met [letters *a*, *b*, and *c* (arrows)]. Letters *d* and *e* (arrows) show excessive patient triggering efforts.

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✓ (Figure 11-31) Letters *a*, *b*, and *c* are examples of respiratory distress where the inspiratory flow demand is high throughout the inspiratory phase. Compare the abnormal near-constant flow patterns (*a*, *b*, and *c*) to the normal descending flow patterns (first flow wave). Letters *d* and *e* show high triggering efforts.

✓ (Figure 11-31) Letters *f* and *g* show that the expiratory flow waves do not return to baseline before the next breath. Incomplete expiration may lead to air trapping.

accompany such a graphic display. The second, third, and fifth flow waves, *a, b*, and *c* (arrows), show that flow demand is high (35 to 45 L/min) throughout inspiration, sustaining near-constant flow wave patterns, which are created under conditions of stress. The flow-time waves do not show a relaxed pattern as in the first flow wave, shown for comparison, where flow gradually descends to cycle the ventilator into expiration at 25% of the initial flow. Instead, flow demand is sustained (squared off) at a high level and ends abruptly, dropping quickly to zero with the patient's expiratory effort and attempts to quickly trigger another breath. The more that flow appears to square off and drop perpendicularly through the flow cycling level, the higher the flow demand, and the less the patient is relaxing during inspiration and pausing between breaths at that set level of pressure support.

The patient's high respiratory drive is also evident by evaluating the patient-triggering efforts in the pressure-time waveforms (*d* and *e* arrows). Negative pressure drops substantially below the sensitivity threshold level set below PEEP before the ventilator demand valve can respond. Air trapping is apparent in the graphic as well, which can be seen on the third and fourth expiratory flow waves (*f* and *g* arrows). Thus, expiration is not complete before the patient triggers breaths four and five. Several of the pressure waveforms are not squared off, so, obviously, the pressure limit set is not being sustained as it should be. Increasing the pressure support level to 15 to 20 cm H_2O would probably eliminate this patient's distress and provide adequate support for a normal level of WOB. If pressure-controlled ventilation (PCV) in the CMV mode were being demonstrated in this example at the same pressure level setting, similar pressure patterns of dyssynchrony would be manifest, but fluctuations in flow to accommodate demand would be less likely. The T_I would be maintained, however, consistent with the settings for PCV in the CMV mode.

USING EXPIRATORY FLOW AND PRESSURE WAVEFORMS AS DIAGNOSTIC TOOLS

Increased Airway Resistance

The expiratory flow waveform can be used to determine whether a patient has excessive airway resistance (asthma), obstructive, or restrictive disease (ARDS). The solid line expiratory flow wave in Figure 11-32 demonstrates excessive expiratory airway resistance. The dashed line represents a relatively normal waveform. Circuit and endotracheal tube (ETT) resistance (i.e., size 6 mm I.D. ETT) can complicate the assessment by creating a similar pattern, but RCPs can easily measure circuit resistance and eliminate it as the cause for the abnormal expiratory flow curve. Typically, circuit resistance is about 6 to 8 cm H_2O/L/sec at 60 L/min with an 8 mm I.D. ETT and humidifier in line (Dennison et al., 1989), which can be subtracted from clinical measurements of airway resistance. The low-peak expiratory flow, the average flow level, and abnormally long expiratory flow time (>5 sec) compared to the normal curve are obvious signs of severely elevated airway resistance in this example. High

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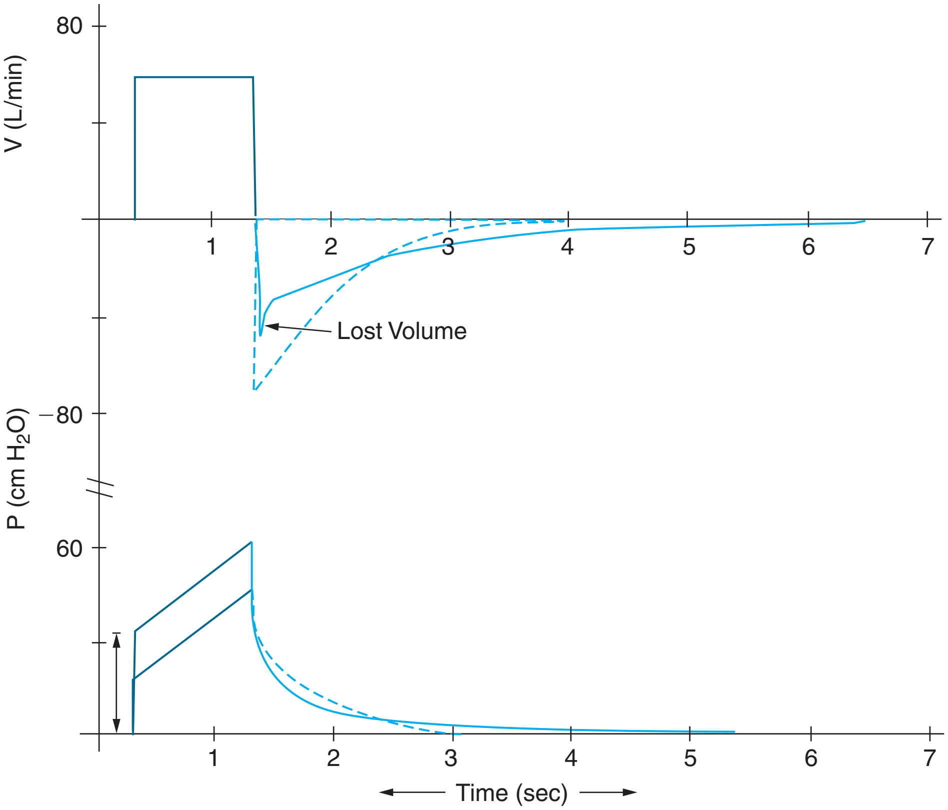

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FIGURE 11-32 The effects of normal airflow resistance (dotted lines) and increased airflow resistance (solid lines) on the expiratory flow and pressure-time waveforms. When the airflow resistance is increased, a lower expiratory flow and a higher peak inspiratory pressure are noted.

resistance will quickly reduce peak expiratory flow rate to low levels compared to normal expiratory flow waves given the same driving pressure (peak P_{ALV}) at end-inspiration. Peak P_{ALV} is usually normal for mandatory V_T (8 to 12 mL/kg ideal body weight) for most obstructive diseases other than emphysema. The spiked initial peak expiratory flow (arrow) indicates the lost volume (compressed gas not delivered to the patient) as it decompresses and is driven from the expiratory limb of the ventilator circuit ahead of lung volume being expired (Nilsestuen et al., 1996), because it is under high ventilating pressures (PIP = 60 cm H_2O).

The solid-line pressure-time waveform represents the pattern that should be expected for the abnormal expiratory flow waveform presented in this example. This is a typical pattern for exacerbated asthma or bronchitis. The initial P_{TA} (double-headed arrow) and PIP (60 cm H_2O) are well above typical levels caused by airway/circuit resistance. The expiratory pressure measured in the expiratory limb of the circuit is lower than normal because expiratory flow is decreased as the V_T is being slowly expired through narrowed airways. Expiratory time (T_E) is increased as a result. The dashed line depicts inspiratory pressure when airway resistance is relatively normal and the P_{TA} and PIP are lower. The circuit expiratory pressure recorded is higher because expiratory flow rate through the circuit is greater and so is flow-resistive pressure as a result. Since T_E is longer than normal for obstructive diseases, there is greater potential for volutrauma

✓ (Figure 11-32) The expiratory flow solid line in the flow-time waveform shows that an increase in airflow resistance (e.g., obstructive lung disease) prolongs the expiratory time. This condition may lead to air trapping, auto-PEEP, or volutrauma.

✓ (Figure 11-32) The *solid* lines in the pressure-time waveform show that an increase in airflow resistance leads to higher PIP, lower expiratory flow, and longer expiratory time.

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from auto-PEEP, which will be readily apparent when graphics are available. A patient may have to be sedated and paralyzed temporarily to be mechanically controlled at low frequencies (6 to 8/min) and low V_T (e.g., 4 to 7 mL/kg IBW) to prevent severe auto-PEEP and lung damage (ARDSNet, 2000).

Loss of Elastic Recoil

✓ (Figure 11-33) The *solid* lines show that in conditions of high compliance or loss of elastic recoil (e.g., emphysema), the peak expiratory flow is decreased (loss of the spiked peak). The PIP is also decreased because less pressure is needed to ventilate lungs with high compliance.

Figure 11-33 shows the waveforms for a patient with emphysema (solid lines of expiratory flow) compared to one with normal lung functions (dashed lines). The expiratory flow wave is similar to the example in Figure 11-32 for the asthmatic/bronchitic patient, but with noted changes. First, the spiked peak expiratory flow is not present because ventilating pressures for emphysema patients are typically low where PIP is 25 cm H_2O (solid-line pressure wave) for a substantially high V_T of 1.0 L being delivered. Also, lung tissue recoil pressure (peak P_{ALV}) and the consequent expiratory flow driving pressure is reduced (approximately 15 cm H_2O in this example since initial P_{TA} is approximately

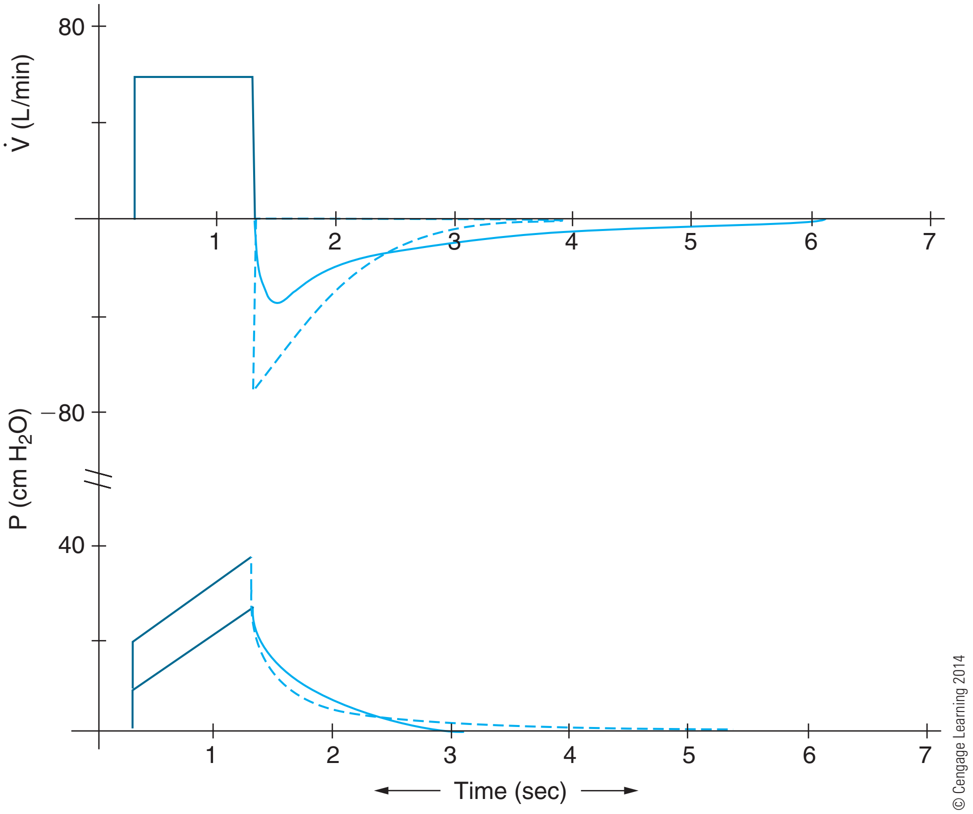

FIGURE 11-33 The effects of normal elastic recoil (dotted lines) and loss of elastic recoil (solid lines) on the expiratory flow and pressure-time waveforms. When the elastic recoil is decreased (i.e., low elastance or high compliance), a lower expiratory flow and a lower peak inspiratory pressure are noted.

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10 cm H_2O), and as a result the peak expiratory flow pattern develops more slowly and is rounded, and T_E is prolonged as demonstrated by the expiratory flow and pressure patterns.

Decreased Lung-Thorax Compliance (C_{LT})

✓ (Figure 11-34) The *solid* expiratory flow curve shows that in conditions of low C_{LT}, the expiratory flow is generally higher and expiratory time is shorter.

✓ In air trapping, the expired volume is less than the inspired tidal volume for one or more breaths. The trapped air comes out with a subsequent mandatory breath resulting in a much larger expired tidal volume.

When airway resistance remains constant, a decreased C_{LT} (e.g., ARDS) will increase the peak expiratory flow rate and shorten the expiratory time. In Figure 11-34, the normal expiratory flow curve (dashed) is compared to the expiratory flow curve in conditions of low C_{LT} (solid lines). The expiratory flow in conditions of low C_{LT} is generally higher and expiratory time is shorter (solid line). A shorter expiratory time correlates with faster lung-emptying in conditions of low C_{LT}.

The pressure waveform (Figure 11-34) shows that the PIP (solid line) would increase as C_{LT} decreased. Note that since the flow rate is the same and airway resistance is unaffected, the initial flow-resistive pressure (arrow) has not changed. Thus, the increase in PIP must be the result of a rise in peak P_{ALV} (plateau pressure). The expiratory portion of the pressure waveform (solid line) generated in the circuit on average is higher under conditions of low C_{LT} and

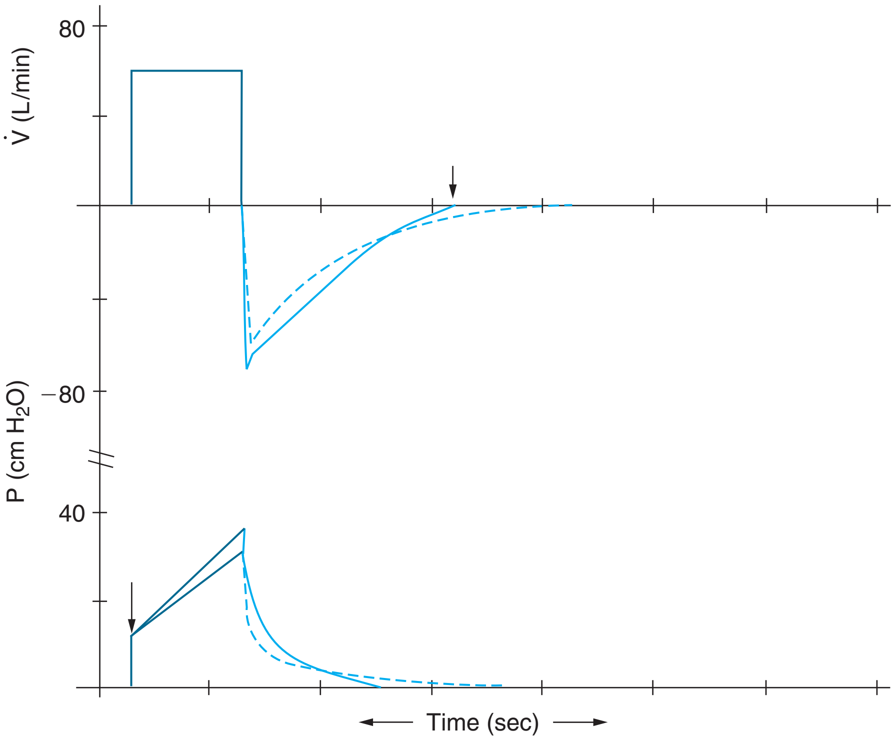

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FIGURE 11-34 The effects of normal compliance (dotted lines) and decreased compliance (solid lines) on the expiratory flow and pressure-time waveforms. When the compliance is decreased, a higher expiratory flow, a shorter expiratory time, and a higher peak inspiratory pressure are noted.

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expiratory time is shorter compared to average conditions for ventilator patients (dashed line).

Gas Trapping and Uncounted Breathing Efforts

Gas trapping can easily be recognized by examining the volume-time waveform. In air trapping, the expired volume is less than the inspired tidal volume (i.e., the expired volume curve does not reach baseline). This observation may occur for one or more breaths. The trapped air typically comes out along with a normal tidal-volume breath resulting in a larger expired tidal volume than inspired tidal volume (i.e., the expired volume curve extends below the baseline). This cycle may repeat as long as air trapping continues.

✓ (Figure 11-35) Double arrow *a* shows that the expiratory flow is dropping rapidly (flow is near zero, parallel to baseline) with a corresponding drop in pressure. This suggests the patient is trying to *inspire* during the expiratory phase.

Air trapping may also be recognized by studying the expiratory flow and pressure waveforms. In Figure 11-35, the expiratory flow in the first flow wave is similar to patterns often observed in clinic for patients with obstructive airway diseases from bronchoconstriction, lesions, or severe excess in airway secretions. The first double-headed arrow *a* indicates that the expiratory flow is dropping rapidly toward zero flow. This may indicate that the patient has a high drive to

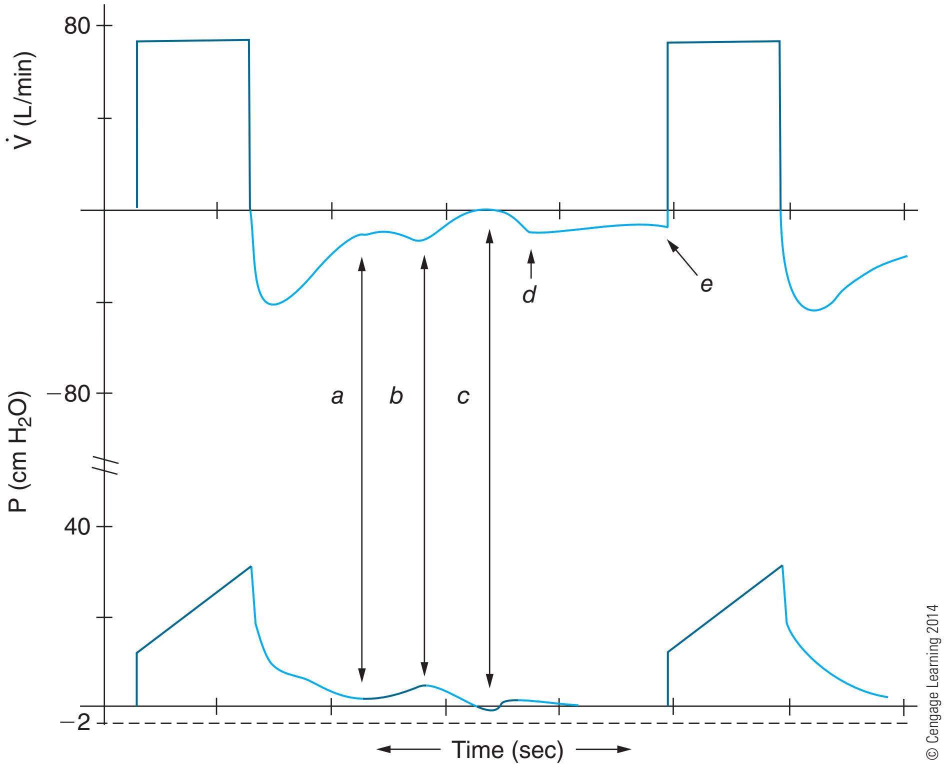

FIGURE 11-35 Expiratory flow and pressure waveforms during volume-controlled ventilation showing airflow obstruction, spontaneous inspiratory efforts, and auto-PEEP during the expiratory phase.

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breathe and is trying to "inspire" during expiration and, hence, is slowing the expiratory flow and reducing expiratory pressure as pointed to (arrow *a*) on the pressure wave below it. It could also indicate that airway obstruction is slowing flow. As a result of hyperinflation or trapped gas some patients cannot expand their thorax or lungs enough to create negative pressure in the ventilator-patient circuit. Their ineffective inspiratory efforts may only slow the flow of gas being released from areas of the lungs, which have longer time constants. They may be able to reduce the driving pressure (P_{ALV}), but may not be able to create sufficient negative P_{AO} to trigger a breath. The drop in flow or pressure may be obstruction to flow, which might be caused by excess secretions, structural damage to airways, or dynamic airway compression. Obstruction from a more homogeneous condition (bronchoconstriction) may present a different pattern (Figure 11-32).

✔ (Figure 11-35) Double arrow *b* shows that the expiratory flow is increasing suddenly with a corresponding rise in pressure. This suggests that the patient is trying to actively *expire* or there is a sudden relief of an obstruction.

The second double-headed arrow *b* points to a sudden increase in flow and corresponding expiratory pressure. This can occur if the patient attempts to actively expire or the obstruction is momentarily relieved with a change in flow dynamics and time constants.

✔ Lack of ventilator response may be caused by dysfunction of the inspiratory valve or inappropriate sensitivity setting.

A third double-headed arrow *c* points to an obvious inspiratory effort during expiration because flow drops momentarily to zero and an insufficient negative pressure (above sensitivity threshold) is recorded. The patient continues to expire more of the trapped gas (arrow *d*) and flow increases and some positive pressure is recorded. A controlled mandatory breath is then time-triggered before the patient has completed expiration as indicated (arrow *e*).

✔ (Figure 11-35) Double arrow *c* shows an inspiratory effort during expiration. Note that the flow momentarily drops to baseline and a negative pressure is recorded.

The patient needs to be assessed for respiratory movements whenever such patterns are observed. V_T and T_I may have to be reduced to allow more T_E, and the sensitivity threshold may have to be increased (-0.5 versus -2 cm H_2O). The respiratory effort needs to be properly documented during evaluation for weaning. Patients often trigger two or three times as many breaths as recorded by the ventilator. Inspiratory efforts during expiration can be felt by placing a hand over the patient's abdomen. Contraction of the abdominal muscles can be felt during forced expirations following the ineffective inspiratory efforts. Visual signs of chest movement, supra- or subclavicular retractions, and interruption of expiratory flow can be heard on auscultation during inspiratory efforts. The graphics can be monitored at the same time to confirm the coordination of efforts with the physical signs of respiration.

TROUBLESHOOTING VENTILATOR FUNCTION

Lack of Ventilator Response

Another use of graphics is to check proper ventilator function. For example, inspiratory valve dysfunction (i.e., a sticking valve) or an out-of-calibration sensitivity threshold can be readily observed when the sensitivity tracing on the pressure-time waveform does not match the sensitivity setting on the ventilator.

Circuit Leaks

(Figure 11-36) Arrow *a* shows air leak since the expiratory volume is less than the inspired tidal volume.

(Figure 11-36) Arrow *b* shows that the air leak (less volume) lowers the PIP—the pressure required for volume delivery.

(Figure 11-37) When a circuit leak occurs in the presence of PEEP, the circuit pressure may drop to or below the preset sensitivity level (dashed lines). This causes autotriggering and rapid mechanical breaths.

Volume waveforms are primarily used to ensure accurate V_T delivery. They can also be used to check for air leak (Figure 11-36). In the second volume waveform, letter *a* (arrow) demonstrates that a leak has developed, since the volume never returns to the zero baseline. The ventilator still delivers the same flow pattern, but the pressure waveform (*b*) shows that the PIP for ventilation has been reduced, since less volume is being delivered to the patient's lungs. Also note that the expiratory flow waves have decreased volumes expired after the leak develops. The third pressure-time waveform (*c*) is used to emphasize that the negative pressure to trigger the sensitivity threshold may appear the same (although sometimes the descent to the sensitivity threshold may be prolonged), but because negative pressure in the circuit is dependent on gas decompression, it will be more difficult for the patient to reduce pressure to the sensitivity threshold if gas can be drawn from the atmosphere.

Figure 11-37 demonstrates the same leak problem as presented in Figure 11-36. However, in this example, approximately 10 cm H_2O PEEP has been added to the circuit and patient's lungs. Again, volume does not return to zero. The PIPs and expiratory flow patterns have also been reduced for the waveforms depicted after the leak appeared. The arrow indicates that once the leak begins, pressure in the circuit starts dropping to the sensitivity setting below the PEEP level set (dashed lines).

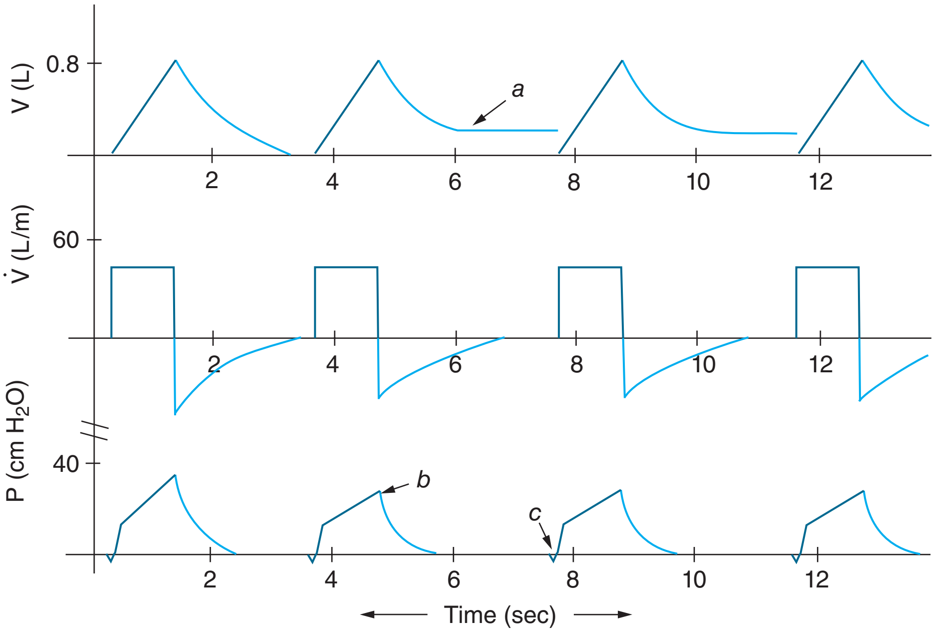

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FIGURE 11-36 Changes to the volume-, flow-, and pressure-time waveforms demonstrate the development of an air leak. Note that the expiratory volume starting from the second breath (*a*) does not return to baseline. The peak inspiratory pressure starting from the second breath (*b*) is reduced from the previous level.

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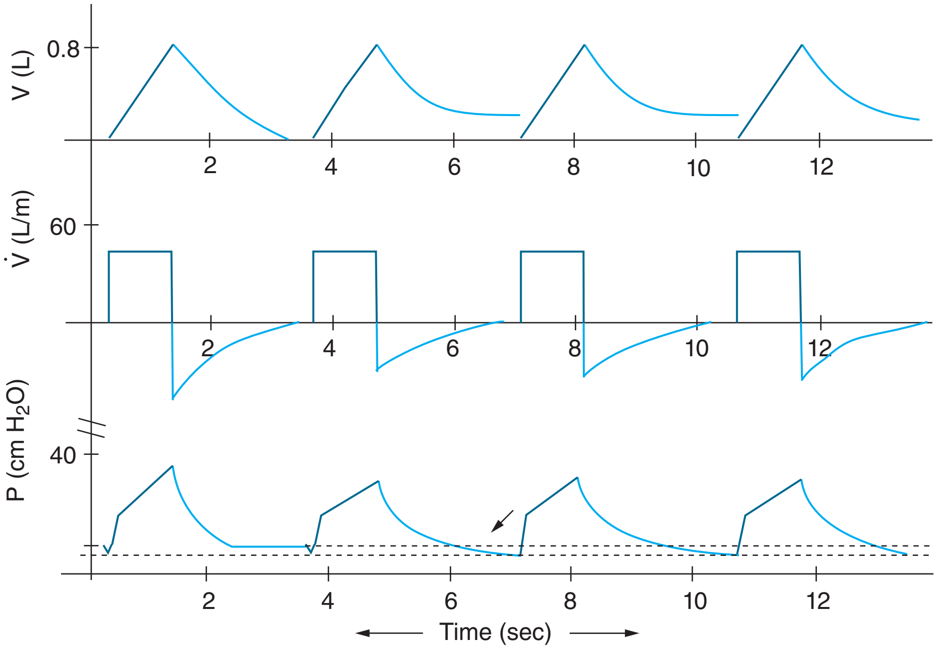

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FIGURE 11-37 Changes to the volume-, flow-, and pressure-time waveforms demonstrate the effect of an air leak when PEEP is used. A reduced circuit pressure (due to air leak) is sufficient to drop the sensitivity level below the PEEP level, causing autotriggering and fast mechanical frequency.

This may lead to autotriggering and go unnoticed unless respiratory frequencies reach extremely high levels (>25 to 30/min).

PRESSURE-VOLUME LOOP (PVL) AND FLOW-VOLUME LOOP (FVL)

Pressure-Volume Loop (PVL)

Another option offered by graphics software is the pressure-volume loop (PVL) presented in Figure 11-38 for an assist breath during constant flow ventilation. The P_{AO} can be read from the *x*-axis and volume from the *y*-axis. The double-headed arrow indicates that the flow-resistive pressure (P_{TA}) is the difference between P_{AO} and P_{ALV} (dashed line). P_{TA} remains the same throughout inspiration, as it does for the step ascending ramp pressure waveform once the initial peak flow is reached and maintained during constant flow ventilation. The PIP of 30 cm H_2O (dashed line) and peak P_{ALV} of 25 cm H_2O are labeled. It is assumed that the C_{LT} is unchanged in this example since P_{ALV} rises linearly with increase in volume.

✓ (Figure 11-38) The double-headed arrow shows the flow-resistive pressure or transairway pressure (P_{TA}). $P_{TA} = P_{AO} - P_{ALV}$ throughout the pressure-volume loop. [Note: end-inspiratory P_{AO} = PIP, end-inspiratory P_{ALV} = peak P_{ALV} or plateau pressure. Refer to Figure 11-4 for review.]

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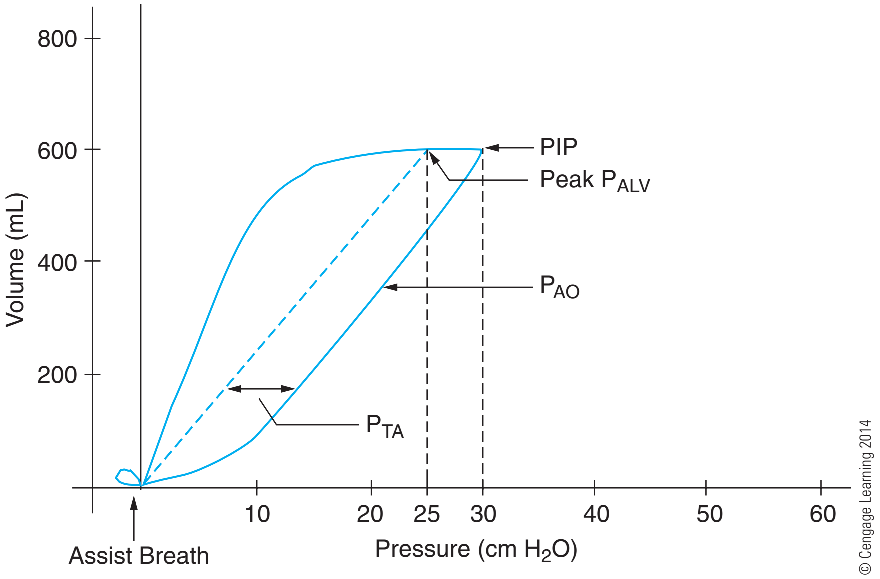

FIGURE 11-38 Characteristics of a pressure-volume loop. The dotted line within the loop is the peak alveolar pressure (P_{ALV} or plateau pressure). The transairway pressure (P_{TA} or flow resistive pressure) is the difference between the P_{AO} and P_{ALV}.

✔ (Figure 11-38) This pressure-volume loop shows that the lung-thorax compliance (C_{LT}) remains unchanged because the P_{ALV} rises linearly with increases in volume.

Effects of Lung-Thorax Compliance on PVL

Figure 11-39 shows a control breath; there is no negative pressure to trigger the mechanical breath. This PVL illustrates how a reduction in C_{LT} causes the loop to move down and to the right (i.e., toward the pressure axis). The increase in

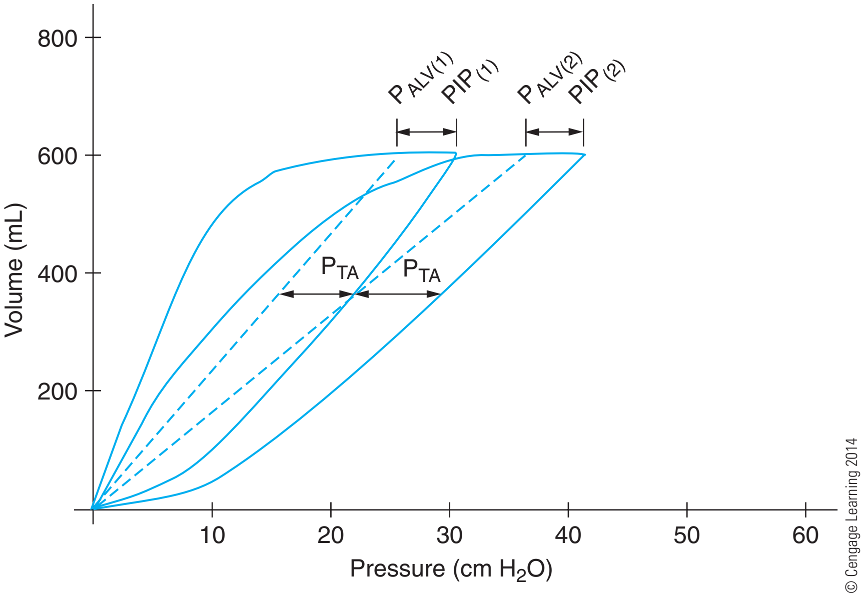

FIGURE 11-39 The effects of lung-thorax compliance (C_{LT}) on the pressure-volume loop during volume-controlled, constant flow ventilation. A decreased C_{LT} shifts the curve toward the pressure axis.

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✔ (Figure 11-39) A decrease in C_{LT} causes the pressure-volume loop to shift toward the pressure axis.

✔ (Figure 11-39) A decrease in C_{LT} will not affect the P_{TA} because the PIP- P_{ALV} gradient (P_{TA}) remains unchanged.

PIP (from 30 to 40 cm H_2O) is proportionate to the increase in P_{ALV} (from 25 to 35 cm H_2O) that is caused by the reduction in C_{LT}. The P_{TA} gradient throughout inspiration (and between PIP and peak P_{ALV}) is held constant. P_{TA} is affected by changes in resistance, not by the changes in C_{LT}.

It is essential to note that on some ventilators (e.g., Servo 300), pressure and volume may be assigned to the *y*- and *x*-axis, respectively. A reduction in C_{LT} would shift the PVL up and to the left (i.e., toward the pressure axis).

Effect of Airflow Resistance on PVL

✔ (Figure 11-40) In situations where the airflow resistance is increased, the P_{ALV} remains unchanged while P_{TA}, PIP, and P_{AO} are increased.

Figure 11-40 shows how the PVL is affected by an increase in resistance (double-headed arrows and dashed lines). P_{ALV} remains unchanged in this example, while inspiratory and expiratory flow-resistive pressure (P_{TA}), PIP, and P_{AO} have all increased.

Lower Inflection Point on PVL and Titration of PEEP

✔ (Figure 11-41) The pressure-volume (compliance) loop shows that the initial point of inflection (Ipi) is the compliance point in which the alveoli are recruited (opened) during mechanical ventilation.

Figure 11-41 shows the effects on the PVL if C_{LT} changes during tidal volume delivery and is presented here only as a point of reference. The dashed line indicates that the slope of the P_{ALV} during V_T delivery has changed. Historically,

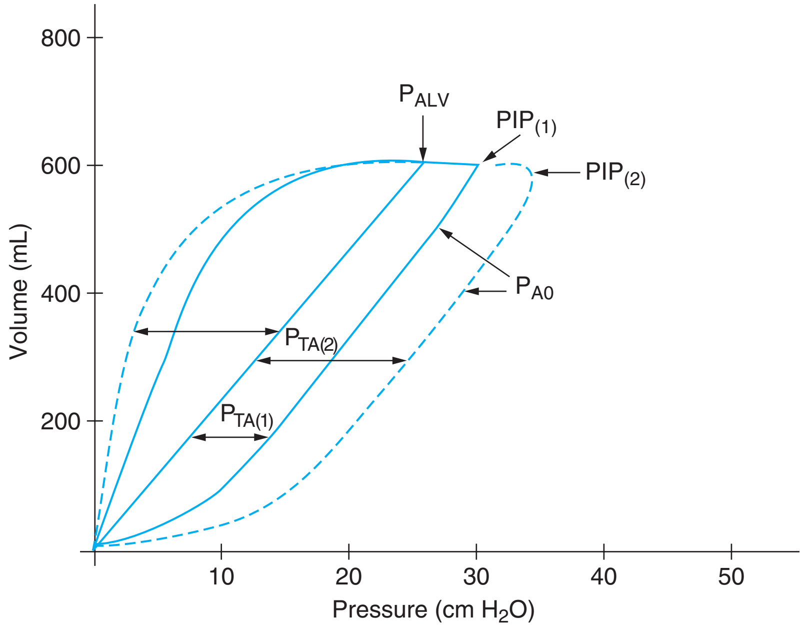

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FIGURE 11-40 The effects of airflow resistance on the pressure-volume loop during volume-controlled, constant flow ventilation. An increased airflow resistance causes increase in PIP, inspiratory and expiratory P_{TA}, and P_{AO}. Note that the P_{ALV} is unchanged.

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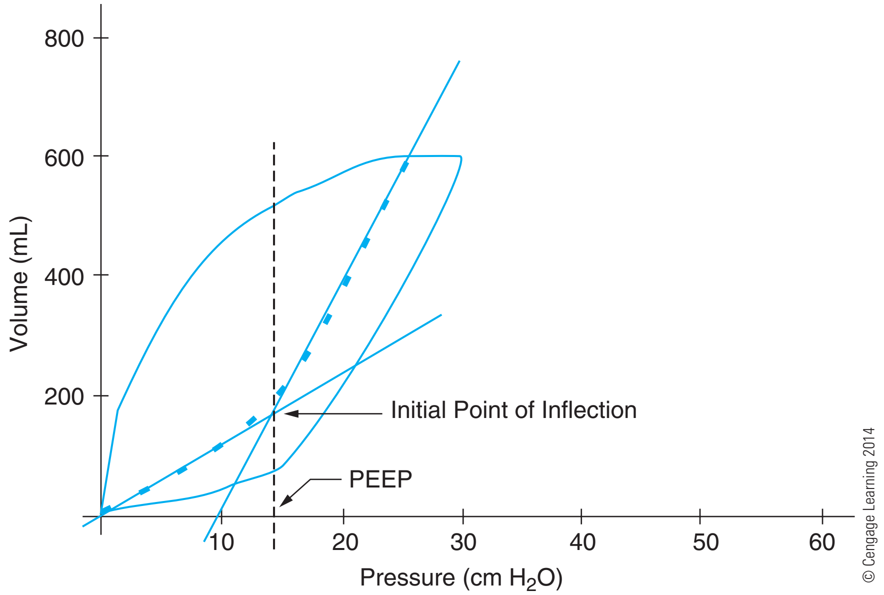

FIGURE 11-41 The initial point of inflection (Ipi) shows the change in the slope from low to improved compliance. A level of PEEP slightly higher (e.g., 2 cm H_2O) than the Ipi may be used to prevent alveolar closure during expiration.

✔ (Figure 11-41) In the presence of Ipi on the pressure-volume loop, PEEP may be added at or slightly above the Ipi to prevent alveoli collapse during expiration.

it was thought that a line could be drawn through the initial slope caused by low C_{LT}, and compared to the line for the slope caused by an improvement in C_{LT}. For lungs with homogenous characteristics (non-ARDS), the change in slope from low to improved compliance is known as the initial point of inflection (Ipi) (Beydon et al., 1991). The Ipi was thought to occur when alveoli were recruited (opened) by volume pressure during inspiration. In this example, P_{ALV} could have been measured by adding progressively larger volumes of gas (beginning at approximately 35 mL) to the patient's lung via a large-volume syringe until the V_T was reached, and then plotting the pause pressures acquired. In the presence of Ipi, some studies suggested that PEEP could be added slightly above (2 cm H_2O) the inflection point to prevent the alveoli from closing during expiration. However, other studies suggest setting PEEP above the closing P_{ALV} pressure on the expiratory side of the curve, to prevent alveolar collapse during mechanical ventilation. Further research has disputed the presence and/or clarity of inflection points being used to set PEEP and usefulness of this strategy in ventilator-patient management. Studies do suggest that the repeated opening and closing of alveoli during mechanical ventilation causes shearing of lung parenchyma (barotraumas), which may promote development of ALI, ARDS, and multiple organ failure.

The Ipi does not apply to lungs with nonhomogenous characteristics (i.e., ALI and ARDS). In ARDS, different lung units have different compliance and opening pressure requirements. For a related discussion, review the sections on ALI, ARDS and recruitment maneuver in Chapter 15.

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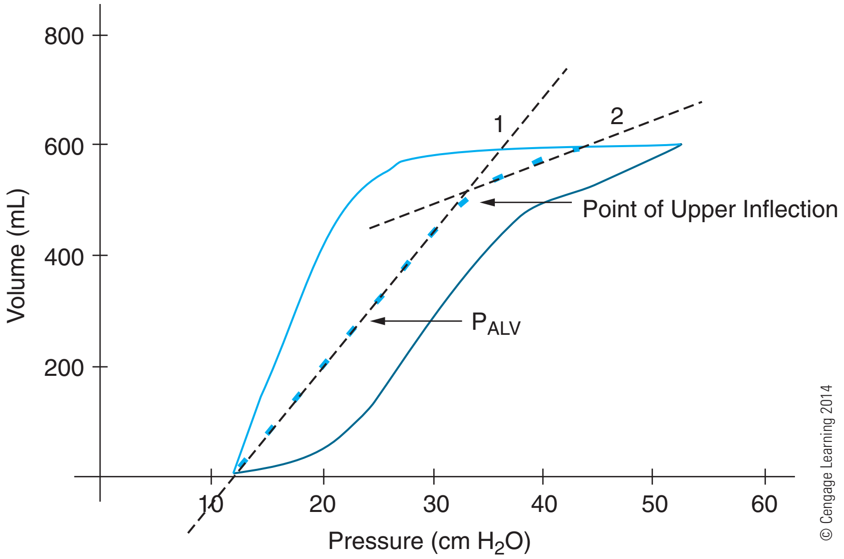

FIGURE 11-42 The point of upper inflection (Ipu) shows a reducing lung-thorax compliance due to hyperdistention of the alveoli. The tidal volume may be reduced until the Ipu (or duckbill) disappears.

Upper Inflection Point on PVL and Adjustment of V_T

✔ (Figure 11-42) In the presence of Ipu on the pressure-volume loop, the tidal volume should be reduced until the Ipu (duckbill) disappears.

✔ (Figure 11-43) On a flow-volume loop, the inspiratory flow is above the horizontal axis, whereas the expiratory flow is below.

✔ (Figure 11-43) Positive response to bronchodilator therapy improves the *expiratory* flow as airflow resistance is reduced. Inspiratory flow is unchanged because it is determined by the settings on the ventilator.

Figure 11-42 presents a PVL where an upper inflection point (Ipu) existed according to early research on this subject. In this example, the P_{ALV} has been plotted as described for Figure 11-37. C_{LT} changed later during V_T delivery because of hyperdistention of the alveoli. The reduction in C_{LT} late in the inspiratory cycle was called the Ipu (Dambrosio et al., 1997). In this example, slope 1 for P_{ALV} (dashed line) is normal, and slope 2 (dashed line) shows a decrease in C_{LT} and the point of upper inflection. The appearance of the upper shape of the P_{AO} curve indicating the presence of an Ipu was known as the duckbill PVL. When a duckbill occurred, the V_T could be reduced until the duckbill vanished (Roupe et al., 1995). Presently, like the Ipi, research is not supporting clear or accurate indications of the Ipu, and it, too, is presented only as a historical reference to issues related to ventilator-patient management of ARDS patients that are in contention and need further study.

Effects of Airway Status on Flow-Volume Loop (FVL)

Figure 11-43 shows another waveform option, the flow-volume loop (FVL). FVLs show flow measurements on the *y*-axis, and volume measurement on the *x*-axis. Inspiratory flow is above the *x*-axis and expiratory flow is below. This example shows two superimposed waveforms during constant flow ventilation at 40 L/min and results of pre- and postbronchodilator therapy. Following bronchodilator therapy, airflow resistance is typically reduced (Garner, 2002).

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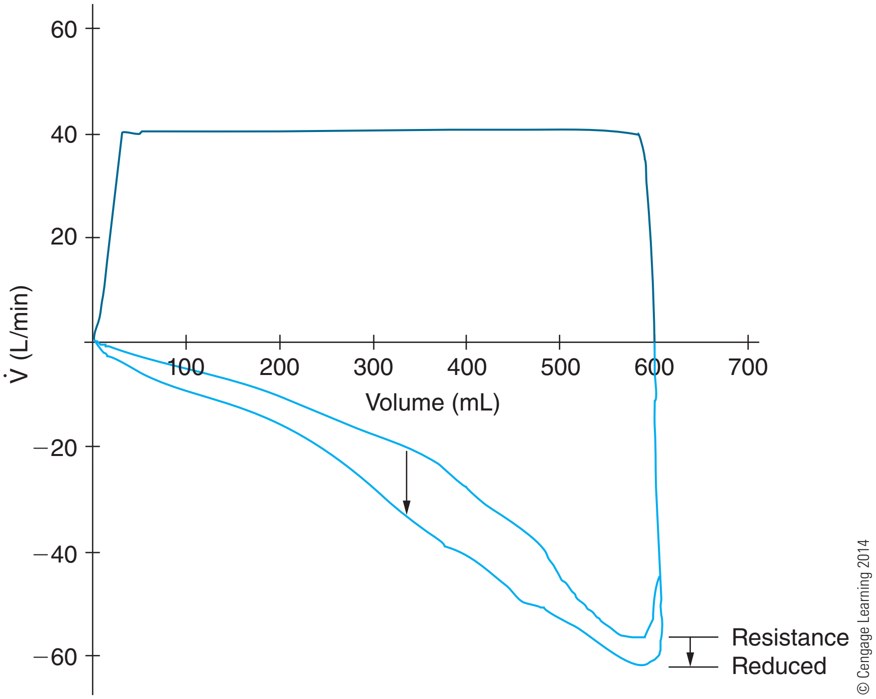

FIGURE 11-43 The effect of airflow resistance on the flow-volume loop. A reduction in airflow resistance increases the peak expiratory flow rate.

SUMMARY

In summary, if graphics of flow-, volume- and pressure-time waveforms, pressure-volume loop, and flow-volume loop could be saved when patients were first placed on a ventilator, ventilator management of patient's progress could be studied and greatly enhanced. Follow-up graphics could be saved and superimposed over the initial copies for comparison, similar to what has been done with the figures throughout this chapter. This can now be done on a minimal level on some ventilator graphics packages. Disk files or printed copies of therapeutic interventions (i.e., bronchodilators, changes in mode of ventilation, chest physical therapy, etc.) and improvements could be documented. Graphic analysis could be used to facilitate discussion and be shared with colleagues and physicians. Outcome assessments of the performance of therapists and respiratory care departments in the area of ventilator management could be documented. A higher quality of ventilator-patient assessment and care is being carried out by practitioners with sufficient expertise in graphics analysis on a day-to-day basis. The sophisticated level of software needed for documentation of these improvements in practice, however, is not readily available. There have been some improvements, but many more are needed to collect and analyze patient clinical status via ventilator waveforms. The technology is available, and it will be exciting to see the eventual changes as the professionals demand further improvements from the ventilator manufacturers. Expertise in waveform analysis at a high level is not required

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for professionals in respiratory care as yet, and the level of expertise is unknown. Those with knowledge and experience know that waveform analysis improves patient care and that it is a very useful tool in ventilator management. However, these improvements need to be studied and supported through research. There is still much to learn. Hopefully the knowledge you gain through study of this work will help serve that purpose.

Self-Assessment Questions

1. The ascending ramp and sine flow waveforms are not used for positive pressure ventilation because the initial flow rate is _______ for most patients. These two waveforms may be appropriate for _______ ventilation.

   A. too high, control
   B. too high, intermittent mandatory
   C. not sufficient, control
   D. not sufficient, intermittent mandatory

2. In volume, pressure and flow waveforms, time in seconds is displayed along the _______ axis.

   A. *x*- or horizontal
   B. *x*- or vertical
   C. *y*- or horizontal
   D. *y*- or vertical

3. Tidal volume can be calculated or determined by measuring the _______ under a _______ waveform.

   A. slope, flow/time
   B. slope, pressure/time
   C. area, flow/time
   D. area, pressure/time

4. The area enclosed under the expiratory flow waveform should _______ the area under the inspiratory flow waveform. If the expiratory volume is less than the inspiratory volume, _______ may be present.

   A. be greater than, airflow obstruction
   B. be equal to, airflow obstruction
   C. be greater than, circuit leak or gas trapping
   D. be equal to, circuit leak or gas trapping

5. In assist/control mode, the I:E ratio is variable because the _______ time of a breath is dependent on the beginning of the _______ breath.

   A. inspiratory, preceding
   B. inspiratory, following
   C. expiratory, preceding
   D. expiratory, following

6. On a pressure waveform, PEEP is present when the end-expiratory pressure rests:

   A. at 0 cm H_2O.
   B. below 0 cm H_2O.
   C. above 0 cm H_2O.
   D. above 5 cm H_2O.

7. On the pressure waveform, assist effort is present when the trigger pressure reaches the _______ setting.

   A. tidal volume
   B. sensitivity
   C. pressure limit
   D. peak flow

8. In CPAP mode, there are no _______ breaths and the airway pressure is above _______ cm H_2O.

   A. mechanical, 0
   B. mechanical, 5
   C. spontaneous, 0
   D. spontaneous, 5

9. An increase in inspiratory flow or airflow resistance would show an unchanged _______ but increased _______. (See Figure 11-11.)

   A. P_{ALV}, PIP and P_{TA}
   B. P_{ALV} and PIP, P_{TA}
   C. P_{TA} and P_{ALV}, PIP
   D. P_{ALV}, P_{TA}

10. A decrease in total compliance (C_{LT}) would show an unchanged _______ but increased _______. (See Figure 11-12.)

    A. P_{ALV}, PIP and P_{TA}
    B. P_{TA}, PIP and P_{ALV}
    C. PIP, P_{ALV} and PIP
    D. P_{ALV}, P_{TA}

11. When the flow waveform selection is changed from constant flow to *true* descending ramp while holding inspiratory time constant, the same volume can only be maintained if the *peak flow* of the descending pattern is:

    A. increased.
    B. decreased.
    C. halved.
    D. doubled.

12. When the flow waveform selection is changed from constant flow to *true* descending ramp during VCV, the same volume can only be maintained if the *inspiratory time* of the descending pattern is:

    A. increased.
    B. decreased.
    C. halved.
    D. doubled.

13. With time-limited ventilation, the higher *initial* peak flow for the descending ramp flow wave creates a _______ initial flow resistive pressure (P_{TA}) than the P_{TA} created by the constant flow. (See Figure 11-14.)

    A. higher
    B. lower
    C. similar
    D. constant

14. With flow-limited ventilation, the initial flow-resistive pressure (P_{TA}) is the same for the _______ flow waves. The *initial* peak flow level stays the same for _______ flow during flow-limited ventilation. (See Figure 11-14.)

    A. constant, constant
    B. constant, descending ramp
    C. descending ramp, descending ramp
    D. constant and descending ramp, constant and descending ramp

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15. In constant flow and descending ramp flow ventilation, the rise in alveolar pressure (P_{ALV}) is directly related to _______ and inversely related to _______.

    A. compliance, volume delivery
    B. compliance, airflow resistance
    C. volume delivery, compliance
    D. volume delivery, airflow resistance

16. At constant T_I, a decreased flow leads to (See Figure 11-15):

    A. higher V_T and P_{ALV}.
    B. higher V_T, P_{TA}, and P_{ALV}.
    C. lower P_{TA} and P_{ALV}.
    D. lower V_T, P_{TA}, and P_{ALV}.

17. During descending ramp flow ventilation, a higher end-flow leads to a _______ V_T and P_{TA} and it _______ affect the P_{ALV}. (See Figure 11-16.)

    A. larger, does
    B. larger, does not
    C. smaller, does
    D. smaller, does not

18. When peak flow is constant (square), _______ are _______ related.

    A. V_T and P_{ALV}; directly
    B. V_T, T_I and P_{ALV}; directly
    C. V_T and P_{ALV}; inversely
    D. V_T, T_I and P_{ALV}; inversely

19. In pressure-controlled ventilation, _______ are typically set by the operator.

    A. rate and I:E ratio
    B. pressure level and I:E ratio
    C. pressure level and rate
    D. pressure level, rate, and I:E ratio

20. In pressure-controlled ventilation, the flow level and V_T delivered are primarily dependent on the:

    A. tidal volume.
    B. tidal volume and lung characteristics.
    C. pressure level set and patient effort.
    D. pressure level set and lung characteristics.

21. During inverse ratio pressure-controlled ventilation, the patients are usually sedated and paralyzed in order to prevent:

    A. barotrauma.
    B. dyssynchrony with the ventilator.
    C. hyperventilation.
    D. hypoxia.

22. In pressure support ventilation, only the _______ level is set and under normal condition, _______ are primarily under the patient's control.

    A. pressure support; flow, volume, and inspiratory time
    B. tidal volume; flow, volume, and inspiratory time
    C. PEEP; flow and volume
    D. plateau pressure; inspiratory time and volume

23. During pressure-controlled ventilation, a(n) _______ airflow resistance or _______ compliance would reduce the delivered flow and tidal volume.

    A. increased, increased
    B. increased, decreased
    C. decreased, increased
    D. decreased, decreased

24. Tachypnea, agitation, accessory muscle use, active expiration, muscle fatigue, and respiratory failure are signs of:

    A. patient-ventilator dyssynchrony.
    B. hyperventilation.
    C. anxiety.
    D. decreased metabolic rate.

25. On a flow waveform, failure of the expiratory flow to return to baseline is indicative of _______ and this condition may lead to _______ and possibly auto-PEEP.

    A. incomplete inspiration, gas trapping
    B. incomplete inspiration, hypoventilation
    C. incomplete expiration, gas trapping
    D. incomplete expiration, hypoventilation

26. In the presence of excessive airway resistance, the expiratory flow is _______ and the expiratory time is _______.

    A. increased, prolonged
    B. increased, shortened
    C. decreased, prolonged
    D. decreased, shortened

27. A decreased C_{LT} leads to a higher expiratory peak flow, a _______ PIP, and a _______ expiratory time.

    A. higher, longer
    B. higher, shorter
    C. lower, longer
    D. lower, shorter

28. A delay of positive pressure waveform (i.e., lack of ventilator response) in spite of a normal negative pressure waveform (i.e., good patient effort) is indicative of:

    A. inadequate line pressure.
    B. ventilator malfunction.
    C. dysfunction of the inspiratory valve or sensitivity setting.
    D. electrical malfunction.

29. Failure of the expiratory flow to return to the zero baseline is indicative of:

    A. gas leak or air trapping.
    B. airflow obstruction.
    C. power failure.
    D. high lung compliance.

30. When a circuit leak occurs in the presence of PEEP, pressure in the circuit drops to the sensitivity setting below the PEEP level and _______ develops and leads to extremely _______ mechanical breaths.

    A. auto-PEEP, slow
    B. auto-PEEP, fast
    C. autotriggering, slow
    D. autotriggering, fast

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31. The difference between P_{AO} and P_{ALV} is: (See Figure 11-38.)

A. PIP.
B. P_{TA}.
C. PEEP.
D. C_{LT}.

32. On a volume-pressure curve, the _______ is assumed to be stable and unchanged if P_{ALV} rises linearly with increases in volume. (See Figure 11-38.)

A. C_{LT}
B. PIP
C. P_{TA}
D. PEEP

33. On a pressure-volume loop, a reduction in C_{LT} causes the loop to move toward the (See Figure 11-39):

A. *y* axis.
B. volume axis.
C. pressure axis.
D. A and B only.

34. On a pressure-volume loop, a reduction in C_{LT} will not change the P_{TA} because the gradient between _______ remains the same. (See Figure 11-39.)

A. P_{TA} and P_{AO}
B. P_{AO} and P_{ALV}
C. PIP and P_{AO}
D. PIP and P_{ALV}

35. On a pressure-volume loop, an increase in resistance would not affect the _______ while the _______ are increased. (See Figure 11-40.)

A. P_{ALV}; P_{TA} and PIP
B. P_{ALV}; P_{TA}, PIP, and P_{AO}
C. P_{TA}; PIP and P_{AO}
D. P_{TA}; P_{AO}, PIP, and P_{ALV}

36. The initial point of inflection (Ipi) occurs when alveoli are recruited during _______. In the presence of Ipi, _______ can be added slightly above the pressure at the inflection point to prevent the alveoli from closing during expiration. (See Figure 11-41.)

A. inspiration, PEEP
B. expiration, PEEP
C. inspiration, tidal volume
D. expiration, tidal volume

37. Overinflation of the alveoli causes a(n) _______ in C_{LT} leading to the appearance of an upper inflection point (Ipu). The Ipu can be minimized by reducing the _______. (See Figure 11-42.)

A. increase, PEEP
B. increase, tidal volume
C. decrease, PEEP
D. decrease, tidal volume

38. On a flow-volume loop, the expiratory flow is _______ the horizontal (volume) axis and it is usually _______ following a successful bronchodilator therapy. (See Figure 11-43.)

A. above, increased
B. above, decreased
C. below, increased
D. below, decreased

Answers to Self-Assessment Questions

1. C.
2. A.
3. C.
4. D.
5. D.
6. C.
7. B.
8. A.
9. A.
10. B.
11. D.
12. D.
13. A.
14. D.
15. C.
16. D.
17. B.
18. B.
19. D.
20. D.
21. B.
22. A.
23. B.
24. A.
25. C.
26. C.
27. B.
28. C.
29. A.
30. D.
31. B.
32. A.
33. C.
34. D.
35. B.
36. A.
37. D.
38. C.

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Additional Resources

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Chapter 12

Management of Mechanical Ventilation

David W. Chang

Outline

- Introduction
- Basic Management Strategies
- Strategies to Improve Ventilation
  - *Increase Ventilator Frequency*
  - *Increase Spontaneous Tidal Volume or Frequency*
  - *Increase Ventilator Tidal Volume*
  - *Other Strategies to Improve Ventilation*
  - *Permissive Hypercapnia*
- Strategies to Improve Oxygenation
  - *Increase Inspired Oxygen Fraction (F_IO_2)*
  - *Improve Ventilation and Reduce Mechanical Deadspace*
  - *Improve Circulation*
  - *Maintain Normal Hemoglobin Level*
  - *Initiate Continuous Positive Airway Pressure (CPAP)*
  - *Initiate Positive End-Expiratory Pressure (PEEP)*
  - *Initiate Inverse Ratio Ventilation (IRV)*
  - *Initiate Extracorporeal Membrane Oxygenation (ECMO)*
  - *Initiate High Frequency Oscillatory Ventilation (HFOV) for Adults*
- Arterial Blood Gases
  - *Respiratory Acidosis and Compensated Metabolic Alkalosis*
  - *Respiratory Alkalosis and Compensated Metabolic Acidosis*
  - *Alveolar Hyperventilation Due to Hypoxia, Improper Ventilator Settings, or Metabolic Acidosis*
  - *Alveolar Hyperventilation in Patients with COPD*
  - *Alveolar Hypoventilation due to Sedation or Patient Fatigue*
  - *Metabolic Acid-Base Abnormalities*
- Troubleshooting of Common Ventilator Alarms and Events
  - *Low Pressure Alarm*
  - *Low Expired Volume Alarm*
  - *High Pressure Alarm*
  - *High Frequency Alarm*
  - *Apnea/Low Frequency Alarm*
  - *High PEEP Alarm*
  - *Low PEEP Alarm*
  - *Auto-PEEP*
- Care of the Ventilator Circuit
  - *Circuit Compliance*
  - *Circuit Patency*

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  Humidity and Temperature
  Frequency of Circuit Change
Care of the Artificial Airway
  Patency of the Endotracheal Tube
  Humidification and Removal of Secretions
  Ventilator-Associated Pneumonia
Fluid Balance
  Distribution of Body Water
  Clinical Signs of Extracellular Fluid Deficit or Excess
  Treatment of Extracellular Fluid Abnormalities
Electrolyte Balance
  Normal Electrolyte Balance
  Sodium Abnormalities
  Potassium Abnormalities
Nutrition
  Undernutrition
  Overfeeding
  Low-Carbohydrate High-Fat Diet
  Total Caloric Requirements
  Phosphate Supplement
Adjunctive Management Strategies
  Low Tidal Volume
  Prone Positioning
  Tracheal Gas Insufflation
Summary
Self-Assessment Questions
Answers to Self-Assessment Questions
References
Additional Resources

Key Terms

alarm
anion gap
auto-PEEP
barotrauma (volutrauma)
brachial plexopathy
culture and sensitivity
extracellular fluid (ECF)
Gram stain
intracellular fluid (ICF)
mechanical deadspace
optimal PEEP
oxygenation
permissive hypercapnia
prone positioning
refractory hypoxemia
spontaneous ventilation
tracheal gas insufflation (TGI)
ventilator-associated pneumonia (VAP)

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Select and use the appropriate strategies to improve ventilation by initiating or altering: ventilator frequency, spontaneous ventilation, ventilator tidal volume, and permissive hypercapnia.
- Select and use the appropriate strategies to improve ventilation by initiating or altering: F_IO_2, mechanical deadspace, circulation, hemoglobin level, CPAP, PEEP, IRV, ECMO, and HFOV.
- Interpret blood gas results based on multiple abnormalities or due to changing patient conditions.
- Troubleshoot and resolve common ventilator alarms and events.
- Provide proper care to the ventilator circuit and artificial airway.

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- Identify the normal values and describe methods to provide normal fluid balance, electrolyte balance, and nutrition.
- Describe the rationale and procedure to initiate: low tidal volume, prone positioning, and tracheal gas insufflations.

INTRODUCTION

The primary function of mechanical ventilation is to support the ventilatory and oxygenation requirement of a patient until such time that the patient becomes self-sufficient. During mechanical ventilation, it is essential to maintain a patient's acid-base balance, nutritional and resting needs, and fluid and electrolyte balance, because these factors can affect management strategies of mechanical ventilation and patient outcome.

This chapter discusses strategies to provide optimal ventilation and oxygenation during mechanical ventilation, as well as other methods to maintain essential physiologic functions through nutritional, fluid, and electrolyte support.

BASIC MANAGEMENT STRATEGIES

The primary goals of mechanical ventilation are to improve ventilation and oxygenation. Essentially all ventilators incorporate designs and features with these two goals in mind. Besides the many modes of ventilation that are available, common settings that are available in most ventilators include frequency (f), tidal volume (V_T), fraction of inspired oxygenation concentration (F_IO_2), positive end-expiratory pressure (PEEP), pressure support ventilation (PSV), and pressure gradient (ΔP). These settings and their intended effects on ventilation and oxygenation are summarized in Table 12-1.

TABLE 12-1 Effects of Ventilator Setting Changes on Ventilation and Oxygenation When Changes Are Indicated

Setting	Ventilation*	Oxygenation**
↑ Frequency (f)	↑↑	↑
↑ Tidal volume (V_T)	↑↑	↑
↑ Fraction of inspired oxygen concentration (F_IO_2)	Unchanged or ↓	↑↑
↑ Positive end-expiratory pressure (PEEP)	Unchanged or ↓	↑↑
↑ Pressure support ventilation (PSV)	↑↑	↑
↑ Pressure gradient (ΔP) (e.g., Bilevel positive-airway pressure, airway pressure release ventilation)	↑↑	↑

** ↑ Ventilation = ↓ $PaCO_2$; ↓ Ventilation = ↑ $PaCO_2$*

*** ↑ Oxygenation = ↑ PaO_2, ↑ SpO_2, ↑ SaO_2*

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STRATEGIES TO IMPROVE VENTILATION

✓ $PaCO_2 > 45$ mm Hg is indicative of hypoventilation (the normal $PaCO_2$ for COPD patients is about 50 mm Hg).

Hypoventilation causes respiratory acidosis (ventilatory failure) and hypoxemia if supplemental oxygen is not provided to the patient. The best measure of a patient's ventilatory status is the $PaCO_2$ level. The normal $PaCO_2$ is 35 to 45 mm Hg; $PaCO_2$ greater than 45 mm Hg is indicative of hypoventilation. For COPD patients, however, the acceptable $PaCO_2$ should be the patient's normal value upon last hospital discharge, and generally it is about 50 mm Hg. When the $PaCO_2$ level goes above this value, significant hypoventilation may be present.

Strategies for improving a patient's ventilation are summarized in Table 12-2.

Increase Ventilator Frequency

The most common approach to improve minute ventilation is to increase the ventilator frequency (f). This may be the control frequency in assist/control, the mandatory frequency in synchronized intermittent mandatory ventilation, or other modes of ventilation that regulate the frequency of the ventilator. However, the ventilator frequency should not exceed 20/min as **auto-PEEP** may occur at or above this frequency, especially during pressure support ventilation (MacIntyre, 1986; Shapiro, 1994). The following

auto-PEEP: Unintentional PEEP associated with pressure support ventilation, high tidal volume and frequency, inadequate inspiratory flow, excessive I-time, inadequate E-time, and air trapping.

TABLE 12-2 Strategies to Improve Ventilation

Priority	Methods
1	Increase ventilator frequency Control frequency in assist/control mode Intermittent mandatory ventilation (IMV) frequency Synchronized IMV frequency
2	Increase spontaneous tidal volume Nutritional support and reconditioning of respiratory muscles Administer bronchodilators Initiate pressure support ventilation (PSV) Use largest endotracheal tube possible
3	Increase ventilator tidal volume Tidal volume in volume-controlled ventilation. Pressure in pressure-controlled ventilation.
4	Reduce **mechanical deadspace** Use low-compliance ventilator circuit Cut endotracheal tube to appropriate length Perform tracheotomy
5	Consider high frequency jet or oscillatory ventilation

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mechanical deadspace: Volume of gas contained in the equipment and supplies (e.g., endotracheal tube, ventilator circuit) that does not take part in gas exchange.

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equations show that an increase in ventilator frequency (f) leads to a higher minute ventilation.

$$\uparrow \text{Minute Ventilation} = (\text{Ventilator } V_T \times \uparrow \text{Ventilator f}) + (\text{Spontaneous } V_T \times \text{Spontaneous f})$$

✔ The most common approach to improve minute ventilation is to increase the respiratory frequency of the ventilator.

It is generally not desirable to increase the ventilator tidal volume beyond a level that is appropriate to the patient's body weight, generally 10 mL/kg (Burton et al., 1997). In volume-controlled ventilation, a larger tidal volume requires a higher peak inspiratory pressure. This high-pressure condition increases the incidence of ventilator-related lung injuries such as cardiovascular impairment and barotrauma.

To estimate the ventilator frequency needed to achieve a certain $PaCO_2$, the following formula may be used, assuming the ventilator tidal volume and deadspace volume stay unchanged (Feihl et al., 1994; Barnes (Ed.), 1994; Burton et al., 1997).

$$\text{New frequency} = \frac{(\text{Frequency} \times PaCO_2)}{\text{Desired } PaCO_2}$$

New frequency:	Ventilator frequency needed for a desired $PaCO_2$
Frequency:	Original ventilator frequency
$PaCO_2$:	Original arterial carbon dioxide tension
Desired $PaCO_2$:	Desired arterial carbon dioxide tension

✔ See Appendix 1 for example.

Increase Spontaneous Tidal Volume or Frequency

In most modes of mechanical ventilation, minute ventilation is the sum of the volume delivered by the ventilator and the volume achieved by a spontaneously breathing patient. For this reason, the patient can contribute to the minute ventilation by increasing either the spontaneous tidal volume or the spontaneous frequency.

$$\uparrow \text{Minute Ventilation} = (\text{Ventilator } V_T \times \text{Ventilator f}) + (\uparrow \text{Spontaneous } V_T \times \uparrow \text{Spontaneous f})$$

It is more advantageous for a patient to increase the spontaneous tidal volume since increasing the frequency usually results in shallow breathing (i.e., rapid shallow breathing pattern) and promotes deadspace ventilation. V_D/V_T ratio is *increased* because of an unchanged anatomic V_D in concurrence with a reduced V_T. ($V_D/\downarrow V_T$ = higher V_D/V_T ratio).

In some patients, the respiratory muscles are not sufficient to maintain prolonged **spontaneous ventilation** or to overcome airflow resistance imposed by the ventilator circuit and endotracheal tube. This condition may be compensated by using pressure support ventilation (PSV). The level of pressure support is usually started at 10 to 15 cm H_2O (Shapiro, 1994) and titrated until a desired spontaneous tidal

spontaneous ventilation: Volume of gas inspired by a patient. It is directly related to the patient's spontaneous tidal volume and frequency.

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volume and frequency are obtained. The increase in spontaneous tidal volume improves the minute ventilation. It is important to note that PSV is only active during spontaneous breathing. PSV is only available in modes of mechanical ventilation that allow spontaneous breathing (e.g., SIMV).

✓ Pressure support ventilation increases spontaneous tidal volume, and therefore the minute ventilation.

Low levels of PSV (<10 cm H_2O) are titrated and used to overcome the airflow resistance of the ventilator circuit and endotracheal tube. At high levels of PSV (>20 cm H_2O), the breathing pattern resembles pressure-controlled ventilation (Burton et al., 1997; Nathan et al., 1993).

$$\uparrow \text{Minute Ventilation} = (\text{Ventilator } V_T \times \text{Ventilator f}) + (\uparrow \text{Spontaneous } V_T \times \text{Spontaneous f})$$

Increase Ventilator Tidal Volume

The ventilator tidal volume is usually set according to the patient's body weight, and its range available for adjustments is rather narrow. Excessive ventilator tidal volume may increase the likelihood of ventilator-related lung injuries. On the other hand, inadequate ventilator tidal volume may lead to hypoventilation and atelectasis.

Before a decision is made to increase the ventilator tidal volume, one must first consider the detrimental side effects of excessive volume and pressure. Increasing the volume should be implemented only when the ventilator frequency is too high and exceeds the patient's ideal breathing pattern and I:E ratio.

Other Strategies to Improve Ventilation

Other strategies to improve the minute ventilation may involve use of ventilator circuits with low compressible volume. This helps to reduce the mechanical deadspace and volume loss due to the circuit internal pressure and tubing compression factor.

The endotracheal tube is sometimes cut shorter to facilitate tube management, to clear secretions, and to reduce deadspace. Tracheostomy also improves ventilation by enhancing tube management and secretion removal. In addition, it provides easier access for oral care and lower deadspace volume than an endotracheal tube.

High frequency jet ventilation has been used primarily in the neonatal population. It is effective to improve ventilation in neonates but its usefulness in adult patients shows mixed results.

Permissive Hypercapnia

permissive hypercapnia: Intentional hypoventilation of a patient by reducing the ventilator tidal volume to a range of 4–7 mL/kg (normally 10 mL/kg). It is used to lower the pulmonary pressures and to minimize the risk of ventilator-related lung injuries. The patient's $PaCO_2$ is significantly elevated and the resulting acidotic pH is neutralized by bicarbonate or tromethamine.

In volume-controlled ventilation, peak inspiratory pressure creates the pressure gradient necessary to deliver a predetermined tidal volume. Occasionally the peak inspiratory pressure can be excessively high in the presence of high airflow resistance and low compliance. This high level of pressure and volume in the lungs may lead to ventilator-related lung injuries.

Permissive hypercapnia is a strategy used to minimize the incidence of ventilator-induced lung injuries caused by positive-pressure ventilation (Hickling,

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2002). Permissive hypercapnia is done by using a low ventilator tidal volume in the range of 4–7 mL/kg (normally 10 mL/kg) (Feihl et al., 1994). The reduced tidal volume lowers the peak inspiratory pressure and minimizes pressure- or volume-related complications. Since the plateau pressure (i.e., end-inspiratory occlusion pressure) is the best estimate of the average peak alveolar pressure, it is often used as the target pressure when trying to avoid alveolar overdistention (Slutsky, 1994). The ventilator tidal volume may be titrated to keep the plateau pressure at or below 35 cm H_2O.

✔ The plateau pressure should be kept below at or 35 cm H_2O to avoid pressure-induced lung injuries.

Low tidal volume may cause hypoventilation, CO_2 retention, and acidosis. Acidosis leads to development of central nervous dysfunction, intracranial hypertension, neuromuscular weakness, cardiovascular impairment, and increased pulmonary vascular resistance. These potential complications may be alleviated by keeping the pH within its normal range (7.35–7.45), either by renal compensation over time or by neutralizing the acid with bicarbonate or tromethamine (Marini, 1993).

Tromethamine (THAM) is a nonbicarbonate buffer that helps to compensate for metabolic acidosis. THAM directly decreases the hydrogen ion concentration and indirectly decreases the carbon dioxide level. The beneficial result is an increased bicarbonate level. Because of its lowering effect on the carbon dioxide level, tromethamine may be preferable to bicarbonate in patients who are being managed with permissive hypercapnia (Kallet et al., 2000). Dosage of 0.3 M tromethamine needed to compensate for metabolic acidosis is calculated by: body weight in Kg × base deficit in mEq/L. Side effects of tromethamine include transient hypoglycemia, respiratory depression, and hemorrhagic hepatic necrosis (Nahas et al., 1998).

✔ Tromethamine (THAM) lowers the carbon dioxide level and increases the bicarbonate levels. It is preferable to bicarbonate in patients undergoing permissive hypercapnia.

By normalizing the pH, it appears that permissive hypercapnia may be a safe and beneficial strategy in the management of patients with status asthmaticus (Cox et al., 1991; Darioli et al., 1984), and adult respiratory distress syndrome (ARDS) (Feihl et al., 1994; Hickling et al., 1990; Lewandowski et al., 1992). The mechanism and physiologic changes of permissive hypercapnia are outlined in Figure 12-1.

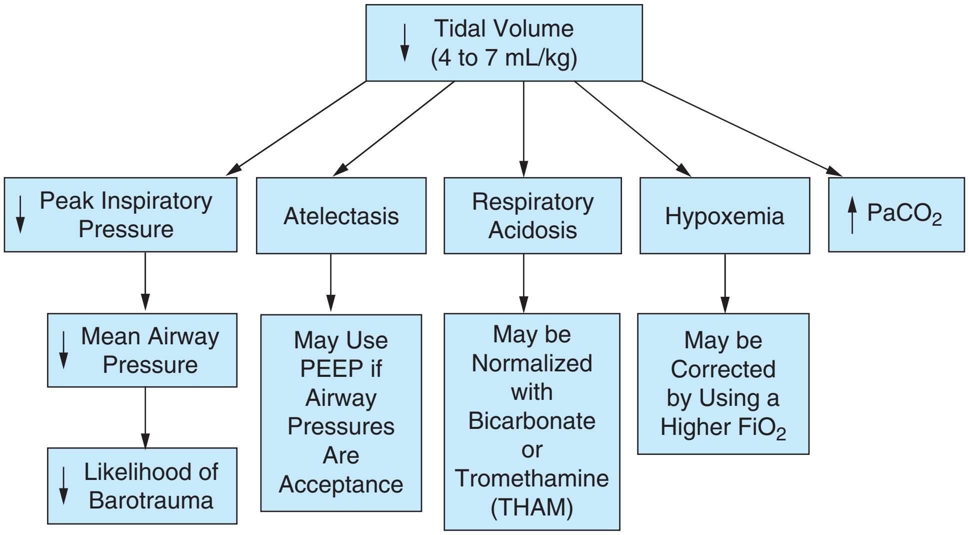
↓ Tidal Volume (4 to 7 mL/kg)
↓ Peak Inspiratory Pressure
Atelectasis
Respiratory Acidosis
Hypoxemia
↑ $PaCO_2$
↓ Mean Airway Pressure
May Use PEEP if Airway Pressures Are Acceptance
May be Normalized with Bicarbonate or Tromethamine (THAM)
May be Corrected by Using a Higher FiO_2
↓ Likelihood of Barotrauma

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FIGURE 12-1 Mechanism and physiologic changes in permissive hypercapnia.

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STRATEGIES TO IMPROVE OXYGENATION

oxygenation: Amount of oxygen available for metabolic functions; affected by ventilation, diffusion and perfusion.

Oxygenation is dependent on adequate and well-balanced ventilation, diffusion, and perfusion. The strategies to improve oxygenation are therefore structured to improve the normal physiologic functions or to compensate for the abnormal ones. The prioritized methods to improve oxygenation, from simple to complex, are outlined in Table 12-3.

Increase Inspired Oxygen Fraction (F_IO_2)

✓ Oxygen readily corrects hypoxemia that is due to uncomplicated V/Q mismatch.

Supplemental oxygen is most frequently used to manage hypoxemia because a high F_IO_2 increases the alveolar-capillary oxygen pressure gradient, thus enhancing diffusion of oxygen from the lungs to the pulmonary circulation. Oxygen readily corrects hypoxemia that is due to uncomplicated V/Q mismatch.

TABLE 12-3 Strategies to Improve Oxygenation

Priority	Methods
1	Increase inspired oxygen fraction (F_IO_2)
2	Improve ventilation and reduce mechanical deadspace
3	Improve circulation Fluid replacement if patient is hypovolemic Vasopressors if patient is in shock Cardiac drugs if patient is in congestive heart failure
4	Maintain normal hemoglobin level
5	Initiate continuous positive airway pressure (CPAP) only with *adequate* spontaneous ventilation
6	Consider airway pressure release ventilation (APRV)
7	Initiate positive end-expiratory pressure (PEEP) Titrate optimal PEEP (See Chapter 15 for titration of optimal PEEP using decremental recruitment maneuver)
8	Consider inverse ratio ventilation
9	Consider prone positioning
10	Consider extracorporeal membrane oxygenation (ECMO), high frequency ventilation, hyperbaric oxygenation

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The following two-step procedure may be used to estimate the needed F_IO_2 for a desired PaO_2 assuming that there is no significant deadspace or shunt abnormalities (Chang, 2012)

$$\text{Step 1: } P_AO_2 \text{ needed} = \frac{PaO_2 \text{ desired}}{(a/A \text{ ratio})}$$

$$\text{Step 2: } F_IO_2 = \frac{(P_AO_2 \text{ needed} + 50)}{713}$$

P_AO_2 needed:	Alveolar oxygen tension needed for a desired PaO_2
PaO_2 desired:	Arterial oxygen tension desired
a/A ratio:	Arterial/alveolar oxygen tension ratio; (PaO_2/P_AO_2 before changes)
F_IO_2:	Inspired oxygen concentration needed for a desired PaO_2
50:	normal $PaCO_2$/Respiratory Quotient = (40/0.8) mm Hg
713:	$P_B - P_{H_2O}$ = (760 − 47) mm Hg

Oxygen and Ventilation. Most patients with respiratory acidosis or ventilatory failure are also hypoxemic. Hypoxemia related to hypoventilation may be partially corrected by improving ventilation. In most cases, supplemental oxygen is also needed for the treatment of hypoxemia. In a clinical setting, an elevated $PaCO_2$ along with hypoxemia should be managed with ventilation and oxygen.

✓ Hypoxemia related to hypoventilation may be partially corrected by improving ventilation. In most cases, supplemental oxygen is also needed to treat hypoxemia.

Oxygen and PEEP. Oxygen therapy alone may not be sufficient if the hypoxemia is caused by intrapulmonary shunting. This type of **refractory hypoxemia** requires oxygen and continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP). CPAP is used for patients with adequate spontaneous ventilation for a sustainable normal $PaCO_2$. PEEP is used for patients requiring mechanical ventilation.

refractory hypoxemia: Hypoxemia that is commonly caused by intrapulmonary shunting and does not respond well to high or increasing F_IO_2.

✓ Refractory hypoxemia responds well to supplemental oxygen when used with CPAP or PEEP. CPAP is used for patients with adequate spontaneous ventilation for a sustainable normal $PaCO_2$. PEEP is used for patients requiring mechanical ventilation.

Oxygen Toxicity. Sufficient oxygen should be given to the patient to maintain a PaO_2 of around 80 mm Hg (lower for COPD patients). Excessive oxygen must be avoided because of the increased likelihood of developing oxygen toxicity, ciliary impairment, lung damage, respiratory distress syndrome, and pulmonary fibrosis (Otto, 1986). Since these complications may occur within 12 to 24 hours of exposure to 100% oxygen, the general guideline is to use an F_IO_2 lower than 60% and limit use of high levels of F_IO_2 for less than 24 hours (Winter et al., 1972).

Improve Ventilation and Reduce Mechanical Deadspace

Adequate ventilation is a prerequisite to oxygenation. Hypoxemia caused by hypoventilation is usually supported by supplemental oxygen during mechanical ventilation, but it must be corrected by improving alveolar ventilation. Arterial $PaCO_2$ is the best indicator of a patient's ventilatory status. When hypoxemia is caused by hypoventilation (i.e., low PaO_2 and high $PaCO_2$), ventilation alone may be sufficient to correct this type of hypoxemia. Ventilation can be provided by increasing the ventilator frequency or tidal volume, or by increasing the patient's spontaneous tidal volume or frequency.

Alveolar ventilation may be improved by ↑ ventilator frequency or V_T or ↑ spontaneous frequency or V_T.

Alveolar ventilation may also be improved by reducing the deadspace volume. Endotracheal intubation and tracheostomy are both effective in reducing the ana tomic deadspace. Mechanical deadspace of an endotracheal tube may be decreased by cutting it shorter than the original length. If a high V/Q mismatch (ventilation in excess of perfusion) exists, alveolar deadspace may be reduced by improving pulmonary perfusion.

Alveolar ventilation may be improved by ↓ the anatomic, mechanical, or alveolar deadspace.

Improve Circulation

Adequate pulmonary blood flow is necessary for proper gas exchange. If perfusion is too low relative to ventilation, deadspace ventilation (high V/Q) results. If perfusion is too high, pulmonary hypertension becomes the potential problem. In order to maintain a normal ventilation-perfusion relationship, the hemodynamic values should be monitored regularly. Hemodynamic monitoring may include invasive procedures such as pulmonary artery catheter and noninvasive procedures such as esophageal Doppler ultrasound and $\dot{V}CO_2$ monitoring.

Hypoperfusion due to congestive heart failure may be corrected by improving the myocardial function.

When hypovolemia occurs due to volume loss, fluid replacement is necessary. If the cause of hypovolemia is shock (i.e., relative hypovolemia; loss of venous tone), fluid replacement should be done with extreme caution because of the potential for fluid overload when vascular tone returns to normal. Vasopressors are useful to provide quick relief from hypovolemia due to shock. The ultimate solution to this type of hypovolemia is to find and correct the causes of shock.

In relative hypovolemia (loss of venous tone), fluid replacement should be done with extreme caution because of the potential for fluid overload when vascular tone returns to normal.

Maintain Normal Hemoglobin Level

Monitoring of the PaO_2 alone for assessment of oxygenation status may be inadequate when a patient's hemoglobin level is below normal. This is because PaO_2 measures the amount of oxygen dissolved in the plasma, whereas a vast majority (>98%) of the oxygen in the blood is combined with and carried by the hemoglobins. During arterial blood gas sampling and analysis, CO-oximetry should be run to evaluate the arterial oxygen content and the hemoglobin levels. Anemia (hemoglobin less than 10 g/100 mL) should be reported along with blood gas results.

Treatment of anemia must be specific to the cause. For example, anemia due to excessive blood loss should be treated by stopping the blood loss and replacing the blood volume. Anemia caused by insufficient hemoglobin should be treated by blood transfusion. Once the hemoglobin level is restored, the arterial oxygen content should return to normal.

Initiate Continuous Positive Airway Pressure (CPAP)

✓ CPAP is only suitable for patients who have adequate respiratory mechanics and can sustain prolonged spontaneous breathing.

Continuous positive airway pressure (CPAP) provides positive airway pressure throughout the spontaneous breathing cycle. It increases the functional residual capacity and is useful to correct hypoxemia due to intrapulmonary shunting. Since CPAP does not provide mechanical ventilation, it is suitable only for patients who have adequate respiratory mechanics and can sustain prolonged spontaneous breathing. Adequacy of spontaneous ventilation can be documented by trending a patient's $PaCO_2$. An increasing $PaCO_2$ over time indicates that the patient is tiring, and continuation of CPAP must be reevaluated.

Initiate Positive End-Expiratory Pressure (PEEP)

✓ CPAP and PEEP increase the functional residual capacity and are useful to correct hypoxemia due to intrapulmonary shunting.

Positive end-expiratory pressure (PEEP) provides positive airway pressure at the end of exhalation from a mechanical breath. It is similar to CPAP with the exception that PEEP is used in conjunction with mechanical ventilation. With PEEP, spontaneous breathing is not required because the patient relies on the ventilator for ventilatory support. Similar to CPAP, PEEP increases the functional residual capacity and is therefore useful to correct hypoxemia due to intrapulmonary shunting.

In order to minimize the cardiovascular complications associated with excessive pulmonary pressures, the **optimal PEEP** should be used in uncomplicated intrapulmonary shunting (e.g., post-operative atelectasis). Optimal PEEP may be determined by evaluating different parameters, such as PaO_2, compliance, O_2 saturation, and ventilator waveforms. Table 12-4 shows that 10 cm H_2O is the optimal PEEP since the next level of PEEP (12 cm H_2O) causes a decrease of PaO_2 and compliance.

optimal PEEP: The lowest PEEP level leading to the best oxygenation status (or other indicators) without causing significant cardiopulmonary complications.

TABLE 12-4 Titration of Optimal PEEP Using PaO_2 and Compliance as Indicators

PEEP (cm H_2O)	PaO_2 (mm Hg)	Compliance (mL/cm H_2O)
0	43	26
5	67	33
8	77	37
10*	83	43
12	79	41

**In this example, 10 cm H_2O is the optimal PEEP since the PaO_2 and compliance show a continuing upward trend with the increasing PEEP level from 0 to 10 cm H_2O. Beyond the optimal PEEP, the next PEEP setting (12 cm H_2O) causes the PaO_2 to drop from 83 to 79 mm Hg and the compliance to fall from 43 to 41 mL/cm H_2O. It is not necessary to use more than one indicator to titrate the optimal PEEP. As shown above, the PaO_2 indicator is more time-consuming and invasive than the compliance indicator. Since compliance is a function of $\Delta V/\Delta P$, the volume-pressure waveform may be used to titrate the optimal PEEP. (See Chapter 15 for titration of optimal PEEP using the decremental recruitment maneuver.)*

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TABLE 12-5 Weaning from PEEP and High F_IO_2

1. Maintain PEEP and decrease F_IO_2 to 40% or 50%	Keep $PaO_2 > 60$ mm Hg or $SpO_2 > 90\%$. Monitor vital signs for acute changes.
2. Maintain F_IO_2 and decrease PEEP to about 3 cm H_2O (at 2 to 3 cm H_2O increments)	Keep $PaO_2 > 60$ mm Hg or $SpO_2 > 90\%$. Monitor vital signs for acute changes.
3. Discontinue PEEP	Monitor vital signs for hypoxia and increased work of breathing.

✔ If the patient is hemodynamically stable and the risk of barotrauma or other PEEP complications appears minimal, it is advisable to wean the F_IO_2 to 40% prior to decreasing the PEEP.

Weaning from PEEP. Since PEEP is used to treat refractory hypoxemia, a patient will typically be receiving high levels of oxygen. The first criterion is to reduce the F_IO_2 to nontoxic levels as quickly as the patient's condition allows. If the patient is hemodynamically stable and the risk of barotrauma or other PEEP complications appear minimal, it is advisable to wean the F_IO_2 to 40% prior to decreasing the PEEP. PEEP should always be decreased in small increments while the patient's oxygen saturation is closely monitored. The oxygen saturation should be kept at or above 90% as this level corresponds to a PaO_2 of 60 mm Hg. The sequence of weaning PEEP is outlined in Table 12-5.

Initiate Inverse Ratio Ventilation (IRV)

Inverse ratio ventilation (IRV) is a technique used in mechanical ventilation in which the inspiratory time is longer than the expiratory time. The inspiratory time is prolonged by decreasing the inspiratory flow rate or by increasing the inspiratory pause time. IRV is also observed during airway pressure release ventilation where the pressure release frequency is less than 20/min (or greater than six seconds per cycle). IRV has been used to treat ARDS patients with refractory hypoxemia not responsive to conventional mechanical ventilation and PEEP (Gurevitch et al., 1986; Morris et al., 1994).

✔ IRV helps to improve oxygenation by (1) overcoming noncompliant lung tissue, (2) expanding collapsed alveoli, and (3) increasing the time for gas diffusion.

The prolonged inspiratory time in IRV helps to improve oxygenation by (1) overcoming noncompliant lung tissues, (2) expanding collapsed alveoli, and (3) increasing the time for gas diffusion. Since inspiratory time is one of the parameters in the calculation of mean airway pressure, a prolonged inspiratory time can increase mean airway pressure and diminish the cardiovascular functions of a critically ill patient.

IRV can be effective in improving oxygenation in patients with ARDS. However, it should be tried on a case-by-case basis and used as an alternative after other conventional mechanical ventilation strategies have failed to improve oxygenation.

Initiate Extracorporeal Membrane Oxygenation (ECMO)

The first use of the extracorporeal membrane oxygenator (ECMO) on an infant was described in 1971 (Zwischenberger et al., 1986). Since then, ECMO has been

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used with considerable success as an oxygenation strategy for infants with severe refractory hypoxemia. In adult patients, however, ECMO has not been shown to provide better oxygenation over conventional mechanical ventilation with PEEP (Zapol et al., 1979).

Initiate High Frequency Oscillatory Ventilation (HFOV) for Adults

High frequency oscillatory ventilation (HFOV) is traditionally used in neonates when conventional ventilation fails to provide adequate ventilation or oxygenation. In recent years, HFOV has been used successfully for the treatment of acute respiratory failure in adult patients based on clinical trials (Viasys Healthcare, 2005).

✓ In HFOV, hypoventilation is managed by using a higher amplitude or a *lower* frequency.

Unlike conventional mechanical ventilation, the $PaCO_2$ is controlled by the power (amplitude) and frequency of oscillation. In HFOV, hypoventilation is managed by using a higher amplitude or a *lower* frequency, and hyperventilation is managed by using a lower amplitude or a *higher* frequency.

Procedure. The following procedure is the suggested clinical guideline based on the 3100B ventilator (Viasys Healthcare, Yorba Linda, CA). The actual application of HFOV must be determined by the physician and based on the patient's condition and requirement (Viasys Healthcare, 2005).

Since the mean airway pressure (mPaw) is affected by the power setting (see next paragraph), the initial mPaw should start at 5 cm H_2O above the mPaw obtained during conventional mechanical ventilation. In patients with severe hypoxia, a mPaw of 40 cm H_2O may be applied for 40 to 60 sec. The mPaw may be increased in 3- to 5-cm H_2O increments every 30 min until the maximum setting. When this strategy is used, oxygenation may worsen in the first 30 min. A chest radiograph should be done within 4 hours to evaluate changes in lung volume.

The power setting determines the amplitude of oscillation and thus the tidal volume. For adult patients, the power is set at 4 and rapidly increased to achieve chest wiggle. Chest wiggle is defined as visible vibration from shoulder to midthigh area. If the $PaCO_2$ rises (with a pH >7.2), the power setting is increased to achieve a change of amplitude in 10 cm H_2O increments every 30 min until it reaches the highest setting.

✓ Unlike conventional mechanical ventilation, a *lower* frequency in HFOV provides a larger tidal volume.

The initial frequency is set at 5 to 6 Hz and may be decreased if unable to control the elevated $PaCO_2$ with amplitude. It is important to note that a lower Hertz setting yields a larger tidal volume. The hertz setting is decreased by 1 Hz increment every 30 min until 3 Hz.

The initial inspiratory time is set at 33% and may be increased up to 50% if unable to ventilate adequately (i.e., by increasing the amplitude or decreasing the frequency). The F_IO_2 is initially set at 100%. The initial settings for ECMO are summarized in Table 12-6.

Weaning from HFOV in Adults. As oxygenation improves, the F_IO_2 is weaned to 40%. Once it reaches 40%, the mPaw is reduced in 2- to 3-cm H_2O increments every

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TABLE 12-6 Initial HFOV Settings for Adults

Parameter	Initial Setting	Note
Mean airway pressure	5 cm H_2O above mPaw obtained during conventional mechanical ventilation	Dependent on power setting.
Power	4	Range 1–10
Controls the amplitude of oscillation (ΔP)		Rapidly increase power to achieve chest wiggle (i.e., visible vibration from shoulder to midthigh area).
Frequency (Hz)	5 to 6 Hz	A *lower* Hz setting yields a *higher* "tidal volume."
Inspiratory time	33%	If unable to ventilate, may increase inspiratory time to 50% by increasing the amplitude or by decreasing the frequency.
F_IO_2	100%	Titrate F_IO_2 as needed.

4 to 6 hours to a 22- to 24-cm H_2O range. At this point, the patients may be switched to pressure control ventilation (PCV) at a frequency of 20 to 25/min, I:E of 1:1, and PEEP of 12 cm H_2O. The pressure setting during PCV is titrated to yield a delivered volume of 6 to 8 mL/kg. The plateau pressure and mPaw should be kept below 35 and 20 cm H_2O, respectively.

ARTERIAL BLOOD GASES

When interpreted correctly, arterial blood gases are very useful in the evaluation of a patient's acid-base, ventilatory, and oxygenation status. Blood gas interpretation is most accurate when it is done in conjunction with the patient's clinical presentation. This section covers two pairs of blood gas abnormalities that look very similar and three blood gas reports that are caused by coexisting conditions: (1) respiratory acidosis and compensated metabolic alkalosis, (2) respiratory alkalosis and compensated metabolic acidosis, (3) alveolar *hyperventilation* due to hypoxia, metabolic acidosis, or improper ventilator settings, (4) alveolar *hyperventilation* in COPD due to hypoxia or improper ventilator settings, and (5) alveolar *hypoventilation* due to sedatives or patient fatigue.

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Respiratory Acidosis and Compensated Metabolic Alkalosis

Respiratory acidosis (ventilatory failure) is caused by hypoventilation. The strategy to correct this abnormality is to improve ventilation. For specific procedures to improve ventilation, refer to the section on “Strategies to Improve Ventilation” at the beginning of this chapter.

✓ If a patient hypoventilates to compensate for metabolic alkalosis, increasing ventilatory support will further compromise spontaneous ventilation.

The strategies to improve ventilation are useful only when respiratory acidosis is caused by hypoventilation. These strategies should not be used when hypoventilation occurs as a compensatory mechanism for metabolic alkalosis. Compensated metabolic alkalosis has an elevated $PaCO_2$, thus mimicking the elevated $PaCO_2$ seen in primary or compensated respiratory acidosis.

Table 12-7 compares the typical blood gases of compensated respiratory acidosis and compensated metabolic alkalosis (both show high $PaCO_2$ and high HCO_3^-). Note that in primary respiratory acidosis, the HCO_3^- is within its normal range (i.e., early stage; no renal compensation). In compensated respiratory acidosis, the pH (7.37) is on the acidotic side of its normal range (7.35–7.45). In compensated metabolic alkalosis, the pH (7.42) is on the alkalotic side of its normal range (7.35–7.45). As with other blood gas abnormalities, the patient’s clinical data and presentation should be used to differentiate a respiratory or metabolic problem.

Respiratory Alkalosis and Compensated Metabolic Acidosis

✓ If hyperventilation is due to metabolic acidosis, reducing the ventilator frequency will cause the patient to continue with hyperventilation until respiratory muscle fatigue occurs.

Respiratory alkalosis is caused by alveolar hyperventilation. In general, this condition does not require mechanical ventilation intervention and it usually allows gradual weaning of the ventilator frequency. However, if the hyperventilation is due to metabolic acidosis, the cause must be identified and treated. Otherwise, weaning the ventilator frequency will cause further patient hyperventilation due to uncorrected and persistent metabolic acidosis.

Additional deadspace tubing between the endotracheal tube and ventilator “Y” adaptor is sometimes used to partially correct persistent respiratory alkalosis. This

TABLE 12-7 Differentiation of Compensated Respiratory Acidosis and Compensated Metabolic Alkalosis

Blood Gas Condition	pH	$PaCO_2$ (mm Hg)	HCO_3^- (mEq/L)
Primary respiratory acidosis	7.31	53	26
Compensated respiratory acidosis	7.38	52	30
Compensated metabolic alkalosis	7.43	50	32

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TABLE 12-8 Differentiation of Compensated Respiratory Alkalosis and Compensated Metabolic Acidosis

Blood Gas Condition	pH	$PaCO_2$ (mm Hg)	HCO_3^- (mEq/L)
Primary respiratory alkalosis	7.51	30	23
Compensated respiratory alkalosis	7.42	27	17
Compensated metabolic acidosis	7.37	25	14

may be necessary when the mechanical volume and frequency cannot be reduced due to the patient's tidal volume and oxygenation requirements.

It is also important to note that hyperventilation (resulting in respiratory alkalosis) is a compensatory mechanism for metabolic acidosis. Compensated metabolic acidosis has a decreased $PaCO_2$, thus mimicking the reduced $PaCO_2$ seen in primary or compensated respiratory alkalosis.

Table 12-8 compares the typical blood gases of compensated respiratory alkalosis and compensated metabolic acidosis (both show low $PaCO_2$ and low HCO_3^-). Note that in primary respiratory alkalosis, the HCO_3^- is within its normal range (early stage; no renal compensation). In compensated respiratory alkalosis, the pH (7.42) is on the alkalotic side of its normal range (7.35–7.45). In compensated metabolic acidosis, the pH (7.37) is on the acidotic side of its normal range (7.35–7.45). The patient's clinical data and presentation should be used to differentiate a respiratory or metabolic problem.

Alveolar Hyperventilation Due to Hypoxia, Improper Ventilator Settings, or Metabolic Acidosis

The blood gas report pH 7.52, $PaCO_2$ 30 mm Hg HCO_3^- 24 mEq/L is typically interpreted as acute respiratory alkalosis. The associated corrective action would be decreasing the ventilator frequency. However, in a mechanically ventilated patient, this type of report can occur if the patient hyperventilates because of persistent hypoxia, improper ventilator settings, or metabolic acidosis. Obviously, action must be taken to find and rectify the underlying causes (e.g., hypoxia). Decreasing the ventilator frequency to correct "respiratory alkalosis" would not be the proper action. In fact, decreasing the ventilator frequency would likely lead to worsening outcomes.

✓ Alveolar hyperventilation (respiratory alkalosis) may occur because of acute hypoxia, improper ventilator settings, or metabolic acidosis.

✓ If hyperventilation is due to persistent hypoxia, reducing the ventilator frequency will cause continuing hyperventilation until respiratory muscle fatigue occurs.

Alveolar Hyperventilation in Patients with COPD

When patients with COPD hyperventilate, the blood gas report may show pH 7.47, $PaCO_2$ 46 mm Hg HCO_3^- 32 mEq/L. The typical interpretation of this report is partially compensated metabolic alkalosis. In reality, this type of blood gas report can occur if the patient with COPD hyperventilates because of acute hypoxia or improper ventilator settings. After correcting the underlying causes, the blood gas

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report would return to the patient's normal pH 7.39, $PaCO_2$ 55 mm Hg HCO_3^- 32 mEq/L (compensated respiratory acidosis).

Alveolar Hypoventilation Due to Sedation or Patient Fatigue

The blood gas report pH 7.30, $PaCO_2$ 50 mm Hg HCO_3^- 24 mEq/L is interpreted as acute respiratory acidosis. The associated corrective action would be increasing the ventilator frequency, tidal volume, or pressure support. However, this type of report can occur if the mechanically ventilated patient hypoventilates because of excessive sedation or respiratory muscle fatigue (e.g., premature weaning attempt). Again, the underlying causes must be found and corrected (e.g., delay weaning). Increasing the ventilator frequency would not be the proper action to correct this "respiratory acidosis."

✓ Ventilatory interventions should not be done to compensate or correct primary metabolic acid-base problems.

✓ Blood gas interpretation *must* correlate with the clinical signs of the patient. Incorrect interpretation can lead to inappropriate changes of ventilator settings or harmful clinical decisions.

Metabolic Acid-Base Abnormalities

Metabolic acid-base abnormalities should be corrected by treating their respective causes. Three major causes of metabolic acidosis are renal failure, diabetic ketoacidosis, and lactic acidosis. One of the major causes of metabolic alkalosis is hypokalemia (Shapiro et al., 1994). Ventilatory (respiratory) interventions should not be done to compensate or correct primary metabolic acid-base problems. The reader should refer to a blood gas textbook for further information on the diagnosis and treatment of metabolic acid-base abnormalities.

Blood gas interpretation *must* correlate with the clinical signs of the patient. Incorrect interpretation can lead to inappropriate changes of ventilator settings or harmful clinical decisions.

TROUBLESHOOTING OF COMMON VENTILATOR ALARMS AND EVENTS

alarm: An absolute value of a parameter on the ventilator beyond which an alert is invoked to warn that the safety limit has been breached.

The type of ventilator **alarm** is easy to spot since most ventilators provide an indicator (light or sound) for each event that triggers the alarm. Once the type of alarm is identified, steps can be taken to alleviate the problem by process of elimination. This section provides the common causes for each alarm.

Low Pressure Alarm

The low pressure limit is set to ensure that a minimum pressure is present in the ventilator circuit during each inspiratory cycle.

Low pressure alarms are triggered when the circuit pressure drops below the preset low pressure limit. If the preset low pressure limit is set at 40 cm H_2O and the circuit pressure drops below 40 cm H_2O, the low pressure alarm will be triggered. In

✓ The low pressure alarm may be triggered in (1) loss of circuit pressure (a common event), (2) loss of system pressure (an uncommon occurrence), (3) conditions leading to premature termination of inspiratory phase, and (4) inappropriate ventilator settings.

all likelihood, the low volume alarm will also be triggered since pressure and volume are affected simultaneously by similar clinical conditions.

Conditions that may trigger the low pressure alarm may be grouped into four areas: (1) loss of circuit pressure (a common event), (2) loss of system pressure (an uncommon occurrence), (3) conditions leading to premature termination of inspiratory phase, and (4) inappropriate ventilator settings. These conditions and selected examples are listed in Table 12-9.

Low Expired Volume Alarm

The low volume limit is set to ensure that the patient receives (and exhales) a minimum volume.

The low expired volume alarm is triggered when the expired volume drops below the preset low volume limit. If the preset low expired volume limit is set at 400 mL, and the expired volume drops below 400 mL, the low volume alarm will be triggered.

✓ The low volume alarm is usually triggered along with the low pressure alarm because loss of airway pressure usually results in loss of volume delivered.

As mentioned before, the low volume alarm is usually triggered along with the low pressure alarm because loss of airway pressure usually results in loss of volume. See Table 12-9 for examples of conditions that may trigger low volume alarm.

TABLE 12-9 Conditions That Trigger the Low Pressure/Low Volume Alarm

Condition	Examples
Loss of circuit pressure	Circuit disconnection Exhalation valve driveline disconnection Endotracheal tube cuff leak Loose circuit connection Loose humidifier connection
Loss of system pressure	Power failure Source gas failure or disconnection Air compressor failure
Premature termination of inspiratory phase	Excessive peak flow Insufficient inspiratory time (I time) Excessive expiratory time (E time) Inappropriate sensitivity setting (too sensitive)
Inappropriate ventilator settings	Excessive frequency with insufficient peak flow Low pressure limit exceeds PIP Low tidal volume limit exceeds V_T

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High Pressure Alarm

✓ The high pressure alarm may be triggered in the following conditions: (1) increase in airflow resistance and (2) decrease in lung or chest wall compliance.

The high pressure limit is set to control the maximum ventilator circuit pressure during a complete breathing cycle, usually during the inspiratory phase.

The high pressure alarm is triggered when the circuit pressure reaches or exceeds the preset high pressure limit. If the high pressure limit is set at 60 cm H_2O, and the circuit pressure reaches or exceeds 60 cm H_2O, the high pressure alarm will be triggered.

Conditions that trigger the high pressure alarm may be (1) increase in airflow resistance and (2) decrease in lung or chest wall compliance. These conditions and examples are shown in Table 12-10.

High Frequency Alarm

✓ The high frequency alarm may be triggered due to (1) the patient's need to increase ventilation and (2) an excessive sensitivity setting.

The high frequency limit is set to alert the practitioner that the patient has experienced tachypnea.

This alarm is triggered when the total frequency exceeds the high frequency limit. Autotriggering of mechanical breaths can trigger the high frequency alarm due to increasing inspiratory effort or incorrect sensitivity setting. Triggering of the high frequency alarm often indicates that the patient is becoming tachypneic-a sign of respiratory

TABLE 12-10 Conditions That Trigger the High Pressure Alarm

Condition	Examples
Increase in airflow resistance	Mechanical Factors Kinking of circuit Kinking of ET tube Blocked exhalation manifold Water in circuit Herniated ET tube cuff Main-stem bronchial intubation High pressure limit set too low Patient Factors Bronchospasm Coughing Patient-ventilator dyssynchrony Secretions in ET tube Biting on ET tube Mucus plug
Decrease in lung or chest wall compliance	Tension pneumothorax Atelectasis ARDS Pneumonia

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distress possibly due to conditions such as hypoxia, pain, anxiety, inadequate inspiratory flow or pressure support. Tachypnia shortens the expiratory time, increases the mean airway pressure, and alters the ventilation/perfusion relationship. For these reasons, persistent tachypnia must be investigated and corrected. Triggering of the high frequency alarm may be corrected by simple steps such as endotracheal suctioning, increasing the F_IO_2, peak flow or pressure support setting. The high frequency alarm limit must not be increased without clear justification (e.g., reversal of sedation or anesthesia).

Another cause of the high frequency alarm is an inappropriate sensitivity setting. When this control is set excessively sensitive to the patient's inspiratory effort, minimum inspiratory efforts or movements will cause the ventilator to initiate auto-triggering and increase in total frequency.

Apnea/Low Frequency Alarm

The apnea/low frequency limit is set to ensure that a minimum number of breaths is delivered to the patient.

✓ Disconnection of the ventilator circuit from the patient's endotracheal tube is the most frequent trigger of the apnea alarm.

The apnea or low frequency alarm is triggered when the total frequency drops below the low frequency limit. Disconnection of the ventilator circuit from the patient's endotracheal tube is the most frequent trigger of the apnea alarm, since the ventilator cannot sense any air movement (respiratory effort) from a disconnected circuit. Other triggers of the apnea/low frequency alarm include a patient under respiratory depressants or muscle-paralyzing agents, conditions of respiratory center dysfunction, and respiratory muscle fatigue.

Some ventilators merely alert the practitioner that the patient is having periods of apnea; the practitioner must increase ventilation to alleviate the situation. Most ventilators switch to a backup ventilation mode until the problem is corrected.

High PEEP Alarm

The high PEEP limit is set to prevent excessive PEEP imposed on the patient. The alarm is triggered when the actual PEEP exceeds the preset PEEP limit. Auto-PEEP may occur in conditions of air trapping, insufficient inspiratory flow (long I-time), or insufficient expiratory time (short E-time).

Air trapping may be reduced by decreasing the ventilator tidal volume and frequency, and by using bronchodilators in patients with reversible airway obstruction. Increasing the inspiratory peak flow provides a shorter I-time and a longer E-time. More time for exhalation helps to reduce air trapping.

Low PEEP Alarm

The low PEEP limit is set to ensure that the preselected PEEP is delivered to the patient. The alarm is triggered when the actual PEEP drops below the preset low PEEP limit. Failure of the ventilator circuit to hold the PEEP is usually due to leakage in the circuit or ET tube cuff.

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Auto-PEEP

✔ Auto-PEEP is associated with pressure support ventilation, significant airway obstruction, high frequency ($>$20/min), insufficient inspiratory flow rates, relatively equal (about 1:1) or inversed I:E ratio, and history of air trapping.

Auto-PEEP (intrinsic PEEP, inadvertent PEEP, occult PEEP) is the unintentional PEEP during mechanical ventilation that is associated with excessive pressure support ventilation, significant airway obstruction, high frequency ($>$20/min), insufficient inspiratory flow rates, and relatively equal (about 1:1) or inversed I:E ratio. It is also more likely to occur when the patient has a history of air trapping (MacIntyre, 1986; Schuster, 1990). With auto-PEEP, the distal airway pressures in the lungs can be as high as 15 cm H_2O, while the ventilator's proximal airway pressure manometer shows zero pressure (or PEEP if PEEP is used). Auto-PEEP can be observed on the pressure-time waveform or measured by occluding the expiratory port just before the next inspiration (Marini, 1988). To measure it accurately, the patient should be sedated or paralyzed.

Auto-PEEP Increases Work of Breathing. Under normal conditions, a mechanical breath is initiated when the inspiratory negative pressure reaches the sensitivity setting of the ventilator. For example, when the normal end-expiratory pressure is 0 cm H_2O and the sensitivity is set at -2 cm H_2O, the pressure gradient (ΔP) or work of breathing to trigger a mechanical breath is 2 cm H_2O (from 0 cm to 2 cm H_2O). See Figure 12-2(A).

If auto-PEEP is present, the work of breathing is increased because the level of auto-PEEP in the lungs at end-expiratory phase must first be overcome before additional inspiratory negative pressure can be used to reach the sensitivity setting. For example, when the auto-PEEP level is 6 cm H_2O and the sensitivity is set at -2 cm H_2O, the pressure gradient (ΔP) to trigger a mechanical breath becomes 8 cm H_2O. Figure 12-2(B) shows the distribution of 8 cm H_2O of pressure (6 cm H_2O to bring auto-PEEP from 6 to 0 cm H_2O plus 2 cm H_2O to reach the preset sensitivity level).

✔ Auto-PEEP may be reduced by reducing the tidal volume or frequency, increasing the inspiratory flow, and eliminating airflow obstruction.

Strategies to Reduce Auto-PEEP. To reduce the likelihood of auto-PEEP, the tidal volume or frequency may be reduced. The frequency during pressure support ventilation should be kept less than 20 breaths per minute if possible. Auto-PEEP may also be minimized or eliminated by improving ventilation or providing a longer expiratory time. Two methods may be useful to reduce or eliminate the auto-PEEP, and they are (1) improving ventilation and reducing air trapping by bronchodilators and (2) prolonging the expiratory time by increasing the flow rate or reducing the tidal volume or frequency.

Using PEEP to Reduce Effects of Auto-PEEP. When setting changes cannot correct auto-PEEP, therapeutic PEEP may be used to reduce the effects of auto-PEEP that is due to air trapping in the small airways (*Note:* patients with fixed obstruction in the large airways should not be managed with therapeutic PEEP). The level of therapeutic PEEP used to counter the effects of auto-PEEP should be kept below 85% of the measured auto-PEEP level (Wilkins et al., 2003). For example, PEEP level of up to 5 cm H_2O may be used when auto-PEEP of 6 cm H_2O is measured during mechanical ventilation. Figure 12-2(C) shows that the pressure gradient (ΔP) to trigger a mechanical breath drops to 3 cm H_2O (1 cm H_2O to bring auto-PEEP from 6 to 5 cm H_2O plus 2 cm H_2O to reach the preset sensitivity level).

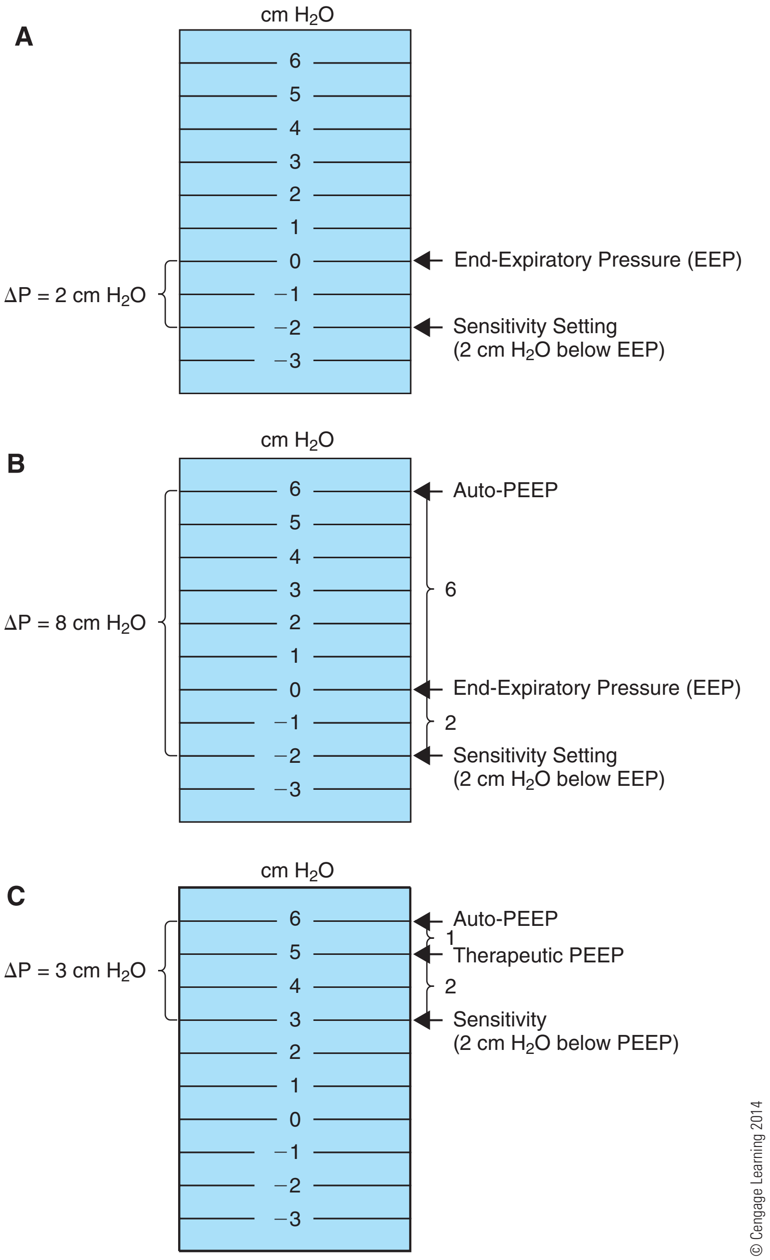
A
cm H2O
6
5
4
3
2
1
0
−1
−2
−3
ΔP = 2 cm H2O
End-Expiratory Pressure (EEP)
Sensitivity Setting
(2 cm H2O below EEP)
B
cm H2O
ΔP = 8 cm H2O
Auto-PEEP
6
End-Expiratory Pressure (EEP)
2
Sensitivity Setting
(2 cm H2O below EEP)
C
cm H2O
ΔP = 3 cm H2O
Auto-PEEP
1
Therapeutic PEEP
2
Sensitivity
(2 cm H2O below PEEP)
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FIGURE 12-2 (A) Without auto-PEEP, the pressure gradient (ΔP) from end-expiratory pressure to sensitivity setting is 2 cm H_2O. (B) With auto-PEEP of 6 cm H_2O, the ΔP from auto-PEEP to sensitivity setting is 8 cm H_2O. (C) With auto-PEEP of 6 cm H_2O and therapeutic PEEP setting of 5 cm H_2O, the ΔP from auto-PEEP to sensitivity setting is 3 cm H_2O.

CARE OF THE VENTILATOR CIRCUIT

The ventilator circuit serves as an important interface between the ventilator and the patient. Circuit compliance, circuit patency, humidity, and temperature are four essential factors in the management of mechanical ventilation.

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Circuit Compliance

The compliance of ventilator circuits should be as low as possible. High circuit compliance leads to a higher compressible volume in the circuit during inspiration, and this condition reduces the effective tidal volume delivered to the patient. For example, at a peak inspiratory pressure of 40 cm H_2O, a ventilator circuit with a compliance of 5 mL/cm H_2O would expand and hold 200 mL (40 cm $H_2O \times 5$ mL/cm H_2O) of the set tidal volume. At the same peak inspiratory pressure, a ventilator circuit with a compliance of 3 mL/cm H_2O would have a compressible volume of only 120 mL (40 cm $H_2O \times 3$ mL/cm H_2O). Unless a tidal volume adjustment is made to account for the circuit compliance factor, the effective (delivered) tidal volume to the patient would be reduced substantially when high compliance circuits are used (Burton et al., 1997).

Circuit Patency

Condensation imposes the most common threat to the patency of ventilator circuits. Gas temperature drops as it travels from the heated humidifier to the patient. As the temperature drops along the circuit, water vapor condenses and water collects in the tubing. This condition leads to significant airflow obstruction. A heated-wire circuit (Figure 12-3) and an inline water trap (Figure 12-4) have been used successfully to reduce condensation and the amount of water in the circuit.

Heat and Moisture Exchanger (HME). Figure 12-5 shows a heat and moisture exchanger (HME) that may be used as a temporary humidification device. The HME is placed between the patient's artificial airway and the ventilator circuit. During exhalation, moisture and heat from the patient are absorbed by the condensation surface of the HME impregnated with $CaCl_2$ or $AlCl_2$. The moisture and heat are transferred back to the patient during the next inhalation. The efficiency of HME units ranges from 70% to 90% relative humidity and 30°C to 31°C (White, 2004). Compared to the heated humidifier, ventilator circuits with a bacterial-viral filtering HME cost less to maintain and are less likely to colonize bacteria (Boots et al., 1997; Kirton et al., 1997).

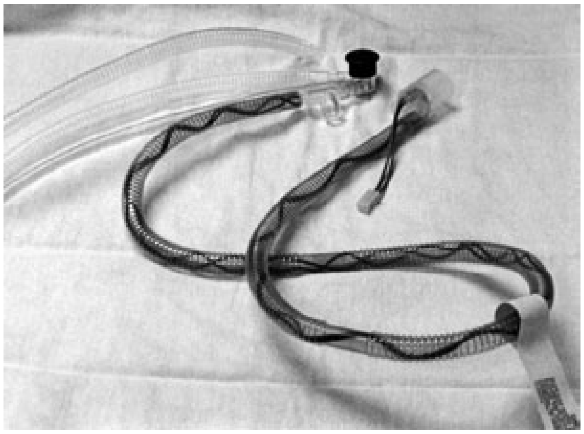
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FIGURE 12-3 A heated-wire circuit.

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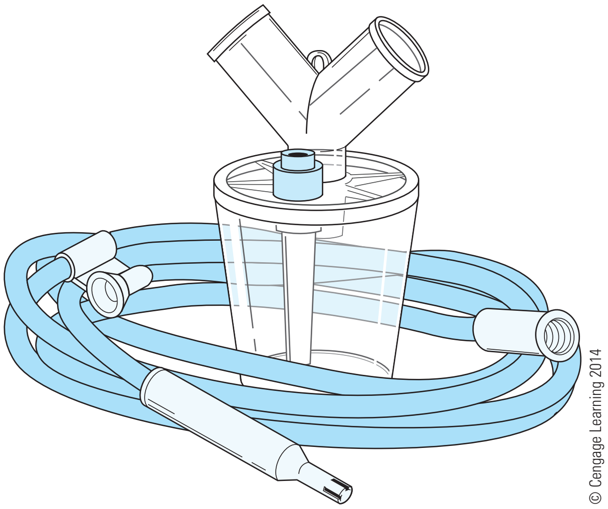
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FIGURE 12-4 In-line water trap.

✔ If a metered-dose inhaler(MDI) is used in conjunction with an HME, the MDI must be placed between the HME and patient.

HME may not be suitable for certain patients due to problems associated with adequacy of airflow, humidity, and temperature. Contraindications for HME include a thick and large amount of secretions, minute volume exceeding 10 L/min, body temperature less than 32°C, and need for aerosolized medications (Wilkins et al., 2003). If metered-dose inhalers (MDI) are used in conjunction with an HME, the MDI must be placed between the HME and patient.

Humidity and Temperature

Since the upper airway is bypassed during mechanical ventilation, the inspired gas temperature should be kept close to the body temperature. The temperature probe

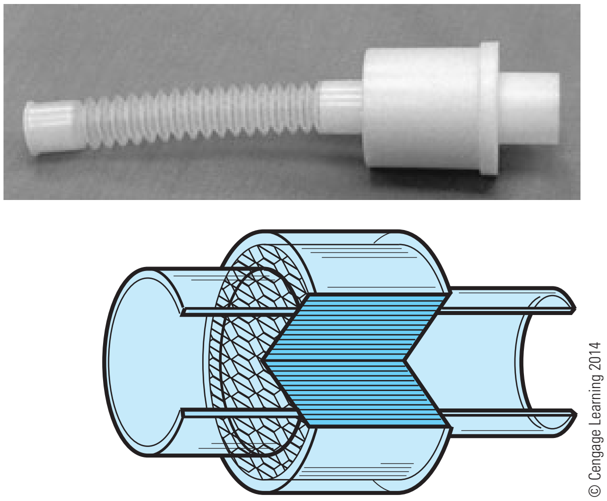
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FIGURE 12-5 A photograph and a functional diagram of a heat and moisture exchanger (HME).

for the heated humidifier should be placed inside the inspiratory limb of the ventilator circuit as close to the patient as possible. Since water vapor saturation depends on the water content as well as the temperature, the temperature setting should be adjusted for a distal temperature reading of 37°C. This ensures proper temperature and humidification to the patient (Burton et al., 1997).

Frequency of Circuit Change

✓ The optimal interval for ventilator circuit change is once per week.

Ventilator circuits should not be changed routinely for infection control purposes. The maximum duration of time that circuits can be used safely is unknown (Hess et al., 2003). For circuits with a humidifier or HME, they should be changed only when visibly soiled (Tablan et al., 2004). Studies have shown that the optimal interval for ventilator circuit change is once per week (Fink, 1998; Kotilainen, 1997; Long et al., 1996; Stamm, 1998). When compared to more frequent circuit changes, weekly circuit change does not increase the incidence of nosocomial infection, including ventilator-associated pneumonia. Weekly change also saves manpower and reduces the direct replacement cost for new ventilator circuits (Kotilainen, 1997).

CARE OF THE ARTIFICIAL AIRWAY

✓ Patency of the ET tube can only be ensured with adequate humidification and prompt removal of retained secretions.

Supplemental humidity must be provided during mechanical ventilation, because the endotracheal (ET) tube does not receive humidification normally provided by the upper airway. In addition, secretions must be removed by suctioning, if necessary, because the ET tube and the ventilator circuit are a closed system. If not removed, any secretions coughed up by the patient are likely to stay in the ET tube. Patency of the ET tube can only be ensured with adequate humidification and prompt removal of retained secretions.

Patency of the Endotracheal Tube

In mechanical ventilation, the primary purpose of an ET tube is to protect the airway and to provide airflow to the lungs. Since airflow resistance is inversely related to the diameter of the tube, small tubes cause a tremendous increase in the work of breathing. In order to maximize airflow, the largest ET tube that is appropriate to the patient should be used. Mucus in the ET tube should also be removed frequently in order to minimize airflow obstruction created by retained secretions.

Poiseuille's Law shows that when the radius of an airway is reduced by half, the driving pressure (work of breathing) must be increased 16 times in order to maintain the same flow rate. An obstructed airway hinders not only mechanical ventilation, but spontaneous ventilation as well. Airway management should always be an integral part of mechancial ventilation.

$$\text{Pressure change} = \frac{\text{Flow}}{r^4}$$

✓ Since the closed suctioning system allows suctioning without disconnecting the ventilator circuit, F_IO_2 and PEEP levels may be maintained.

Other conditions that can affect the patency of the ET tube include (1) kinking or bending of the tube due to poor positioning of the patient and placement of the ventilator circuit; (2) patient biting on the ET tube due to physical or psychologic discomfort; and (3) malfunction of the ET tube cuff causing partial or complete blockage.

Frequent endotracheal suctioning is sometimes necessary to maintain the patency of the endotracheal tube. One of the problems with endotracheal suctioning is hypoxia. Suction-induced hypoxia may be minimized by preoxygenating the patient prior to suction, limiting the total suction time to no more than 10 sec, and using a closed inline tracheal suction system (Wilkins et al., 2003). Since the closed suctioning system allows suctioning without disconnecting the ventilator circuit, F_IO_2 and PEEP levels may be maintained. Closed inline suction catheters may be changed weekly (instead of daily) with no significant increase in the frequency of ventilator-associated pneumonia (Stoller et al., 2003). Figure 12-6 shows a closed tracheal suction system.

Humidification and Removal of Secretions

✓ Instilling a saline solution directly into the airway for the purpose of thinning the secretions or stimulating a cough is not supported by the literature (Branson, 2007).

Proper function of the ciliary blanket of the airway is dependent on adequate humidity. In mechanical ventilation, humidification is commonly provided by a heated humidifier, heated wire circuit, or, for short-term use, a heat and moisture exchanger (HME, or artificial nose). Occasionally, humidification and removal of the secretions are supplemented by use of a saline solution or mucolytic agent via a small volume nebulizer. Instilling a saline solution directly into the airway for the purpose of thinning the secretions or stimulating a cough is not supported by the literature (Branson, 2007).

Saline solution used in a small volume nebulizer is delivered in an aerosol form, and is capable of carrying pathogens into the lower airways. Instillation of saline solution directly into the trachea to facilitate endotracheal suctioning has also been implicated in the contamination of the lower airways with pathogens (Hagler et al., 1994). For these reasons, aseptic techniques for equipment handling and sterile techniques for endotracheal suctioning must be followed in order to minimize the occurrence of pulmonary contamination and ventilator-associated pneumonia (Sole et al., 2003).

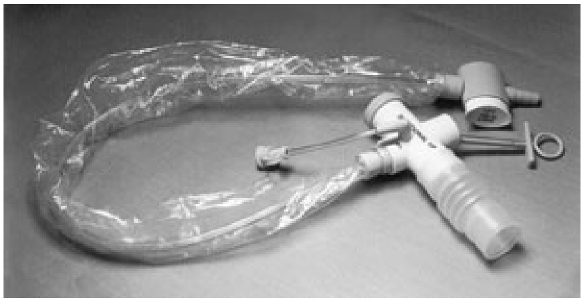

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FIGURE 12-6 An example of a closed suction system manufactured by Ballard Medical, Inc.

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Ventilator-Associated Pneumonia

Patients who are intubated and on mechanical ventilation are more prone to develop nosocomial pneumonia than nonintubated patients (Craven et al., 1989). The estimated incidence of **ventilator-associated pneumonia (VAP)** ranges from 10% to 65%, with fatality rates of 13% to 55% (Kollef et al., 1994). The presence of an artificial airway bypasses the natural defense mechanism of the airway, causes local trauma and inflammation, and increases the risk of aspiration of pathogens from the oropharynx.

ventilator-associated pneumonia (VAP): Infection of the lung parenchyma that is related to any or multiple events that the patient undergoes during mechanical ventilation.

In one study, 45% of the patients developed pneumonia within 3 days of intubation (Lowy et al., 1987). This condition may be caused by microbes acquired from the patient's oropharynx, respiratory instruments, health care providers (Hu, 1991), endotracheal and nasogastric tubes (Joshi et al., 1993), and manual ventilation bags (Weber et al., 1990). Table 12-11 outlines the potential sources of ventilator-associated pneumonia. Strategies to decrease ventilator-associated pneumonias include proper handwashing techniques, closed suction systems (Figure 12-6), continuous-feed humidification systems, change of ventilator circuit only when visibly soiled, and elevation of head of bed to 30° to 45° (Tablan et al., 2004).

For the diagnosis and treatment of VAP, early microbiologic examinations are recommended to guide the use of appropriate antibiotics. Diagnosis and treatment recommendations are beyond the scope of this chapter. Readers should research current publications on VAP and read the articles by Rello et al. (2001) and Koenig et al. (2006). Chapter 15 provides a more detailed discussion on VAP.

Sputum Culture. Sputum cultures should be obtained if infection of the lungs is suspected. Since the patient is intubated, the sputum sample may be obtained via an endotracheal suction setup and a sputum trap (Figure 12-7). Sputum analyses are commonly done by the **Gram stain,** and the **culture and sensitivity** methods.

Gram stain: A method for staining bacteria. Gram-positive bacteria (e.g., *Staphlococcus*) retain the gentian violet (purple) color and gram-negative bacteria (e.g., *Pseudomonas*) take the red counterstain.

culture and sensitivity: A laboratory procedure that grows the microbes in a medium and tests their sensitivity or resistance to different antimicrobial drugs.

TABLE 12-11 Potential Sources of Ventilator-Associated Pneumonia

Potential Source	Locations
Patient	Oropharynx
Health care provider	Hands
Equipment and supplies	Respiratory instruments Aerosol nebulizers and humidifiers Endotracheal tube Nasogastric tube Manual ventilation bag

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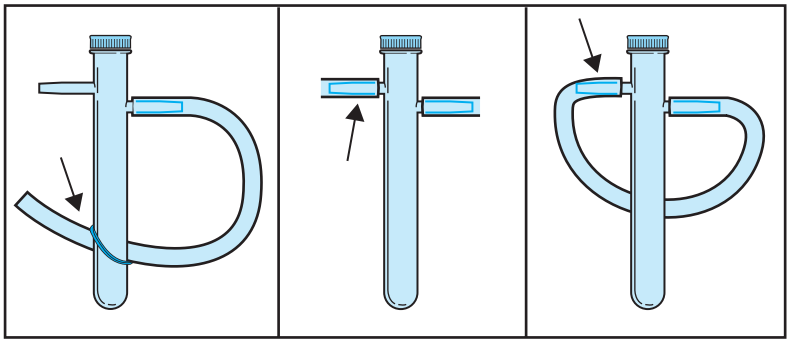

1.
Remove
rubber band.

2.
Place tubing from
suction machine on
upper arm of aspirating
tube. Collect specimen.

3.
After specimen collection,
seal aspirating tube by
placing free end of rubber
tubing on upper arm.
Specimen is ready for lab.

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FIGURE 12-7 Sputum collecting tube used with a suction system. The upper outlet goes to the vacuum source and the lower outlet is connected to the suction catheter.

The Gram stain technique is done to quickly establish the general category (gram-positive or gram-negative) of the suspected microbes so that broad-spectrum antibiotics based on Gram-stain type may be administered without delay. Acid-fast sputum analysis is for pulmonary tuberculosis and silver stain is for *Pneumocystis jiroveci* pneumonia. Culture and sensitivity is more time-consuming, but it can identify the microbes and the most suitable antibiotics for the infection.

In cases where clinical presentations of an infection point to the most likely pathogen, empiric antibiotic therapy may be started without Gram-stain or culture and sensitivity study.

✓ Empiric drug therapy is done without confirmation of the pathogen causing the infection.

FLUID BALANCE

Fluid balance in the body is mainly affected by (1) the blood and fluid volume in the blood vessels and cells, (2) the pressure gradient between the blood vessels and the tissues around them, and (3) electrolyte concentrations.

Distribution of Body Water

Water makes up about 60% of the body weight. The distribution of this volume is 20% in the plasma and interstitial fluid **(extracellular fluid,** or **ECF)** and 40% within the cells **(intracellular fluid,** or **ICF)**. Table 12-12 shows the distribution of body water.

extracellular fluid (ECF): Fluid in the plasma and interstitial space. It accounts for 20% of total body water and is mainly affected by the sodium concentration in the plasma.

intracellular fluid (ICF): Fluid within the cells. It accounts for 40% of total body water.

Changes in Extracellular Fluid Distribution. The distribution of body water in the ECF and ICF compartments is not a static measurement. Depending on the physiologic needs, fluid can move into and out of any compartment along with certain electrolytes. When an excessive volume of fluid moves out of the extracellular compartment, ECF deficit occurs.

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TABLE 12-12 Distribution of Body Water

Compartment	Subdivision	Percent of Water by Body Weight
Extracellular	Plasma	5%
	Interstitial fluid	15%
Intracellular	Intracellular fluid	40%
Total		60% of body weight

Fluid deficiency in the extracellular compartment (space) may be caused by one or a combination of these reasons: (1) inadequate intake (e.g., dehydration); (2) excessive loss (e.g., diarrhea); and (3) shifting of fluid along with the electrolytes into cells and tissues (e.g., swelling of tissues in burns).

Clinical Signs of Extracellular Fluid Deficit or Excess

✔ When urine output drops below 20 mL/hour, it is indicative of fluid inadequacy.

Urine output is the most common method in the assessment of ECF abnormalities. When urine output drops below 20 mL/hour (or 400 mL in a 24-hour period, or 160 mL in 8 hours), it is called oliguria and is indicative of fluid inadequacy (Kraus et al., 1993). Excessive urine output is one of the signs of excessive ECF or excessive diuresis. Other clinical signs of ECF abnormalities include those involved with the central nervous system and the cardiovascular system. They are listed in Table 12-13.

TABLE 12-13 Signs of Extracellular Fluid (ECF) Deficit or Excess

System	ECF Deficit	ECF Excess
CNS	Diminished sensorium Coma	None
Cardiovascular	Tachycardia Hypotension Cold extremities Poor peripheral pulse	Increased pulmonic P_2 heart sound Increased cardiac output Bounding pulse Pulmonary edema
Renal	Oliguria Anuria (no urine)	Increased urine output

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Treatment of Extracellular Fluid Abnormalities

✓ Decrease in heart rate, increase in blood pressure and urine output are signs of improvement in ECF deficit after fluid replacement.

Treatment of ECF deficit is by fluid replacement with Ringer's lactate solution since it is similar to ECF in composition. Physiologic (0.9%) saline solution is an acceptable alternative. Success of fluid replacement therapy can be determined by reversal of those signs of ECF deficit in Table 12-13. For example, decrease in heart rate, increase in blood pressure and urine output are signs of improvement in ECF deficit after fluid replacement.

Excessive fluid in the extracellular space is uncommon in a clinical setting. When it occurs, pulmonary edema is a common manifestation. The treatment for excessive ECF is to withhold fluid or to give a diuretic such as furosemide (Lasix). Mannitol should not be given for diuresis as it can increase plasma volume before inducing diuresis (Eggleston, 1985).

✓ Disappearance of pulmonic P_2 heart sound, reduction in pulse intensity, and clearing of pulmonary edema are signs of improvement in ECF excess.

Use of diuretics will further increase the urine output. For this reason, reversal of the cardiovascular signs of ECF excess in Table 12-13 should be used to determine the success of treatment. For example, disappearance of the pulmonic P_2 heart sound, reduction in pulse intensity, and clearing of pulmonary edema are signs of improvement in ECF excess due to fluid restriction or diuresis. Since diuresis can affect the electrolyte composition, monitoring of electrolyte balance is essential when diuretics are used to manage ECF excess.

ELECTROLYTE BALANCE

Electrolyte balance is the difference between the cations (positively charged ions) and the anions (negatively charged ions) in the plasma. Serum cations and anions are used to calculate the anion gap and assess a patient's electrolyte balance.

Normal Electrolyte Balance

Table 12-14 shows the normal values for serum electrolytes. Sodium is the major cation in the extracellular fluid compartment and it is directly related to the fluid level in the body. Potassium is the major cation in the intracellular fluid compartment and it is not related to the amount of fluid in the body.

Sodium and potassium are the two major electrolytes that must be monitored. In general, once the sodium and potassium concentrations are properly managed and returned to normal, the chloride concentration will be corrected as well without further intervention. The following sections cover sodium and potassium abnormalities.

anion gap: The difference between cations (positive ions) and anions (negative ions) in the plasma. The normal range is 15–20 mEq/L when K^+ is included in the calculation (10–14 mEq/L when K^+ is excluded).

Anion Gap. **Anion gap** is the difference between the cations [sodium (Na^+) and potassium (K^+)] and the anions [chloride (Cl^-) and bicarbonate (HCO_3^-)]. The normal range is 15–20 mEq/L when K^+ is included in the calculation (10–14 mEq/L when K^+ is excluded). When the anion gap is outside this range, electrolyte replacement may be necessary. See Chapter 9 for a discussion on the interpretation of anion gap in metabolic acidosis.

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TABLE 12-14 Normal Serum Electrolytes

Cation	Concentration (mEq/L)	Anion	Concentration (mEq/L)
Na^+	140 (138 to 142)	Cl^-	103 (101 to 105)
K^+	4 (3 to 5)	HCO_3^-	25 (23 to 27)
Ca^{++}	5 (4.5 to 5.5)	Protein	16 (14 to 18)
Mg^{++}	2 (1.5 to 2.5)	HPO_4^{--}, $H_2PO_4^-$	2 (1.5 to 2.5)
		SO_4^{--}	1 (0.8 to 1.2)
		Organic acids	4 (3.5 to 4.5)
Total cations	151	Total anions	151

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✔ See Appendix 1 for example.

The anion gap is calculated as follows:

$$\text{Anion Gap} = Na^+ + K^+ - Cl^- - HCO_3^-$$

or

$$\text{Anion Gap} = Na^+ - Cl^- - HCO_3^-$$

Sodium Abnormalities

Sodium is the major cation in the extracellular fluid (ECF) and it directly influences the ECF volume. The sodium concentration in the ECF may be higher than normal (hypernatremia) or lower than normal (hyponatremia). The clinical signs of sodium abnormalities are highlighted in Table 12-15.

TABLE 12-15 Clinical Signs of Sodium Abnormality

System	Hyponatremia	Hypernatremia
Central nervous system	Muscle twitching Loss of reflexes Increased intracranial pressure	Restlessness system Weakness Delirium
Cardiovascular	Blood pressure change secondary to increased intracranial pressure	Tachycardia Hypotension (if severe)
Gastrointestinal	Watery diarrhea	None
Renal	Oliguria to anuria	Oliguria

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Hyponatremia is commonly related to ECF deficits (hypovolemia). The usual treatment is replenishment of sodium with saline solution.

Hyponatremia. Hyponatremia is a more common form of sodium abnormality than hypernatremia. It is commonly related to ECF deficit (hypovolemia). The usual treatment is replenishment of sodium with saline solution (100 to 300 mL of 2.5% or 3% saline). It is not safe to administer fluids that have no sodium because water intoxication may occur. Rapid movement of sodium-free fluid into the brain cells and kidney cells by the action of osmosis may cause edema and shutdown of these organs (Eggleston, 1985).

Hypernatremia is an uncommon problem and it is usually related to water deficit as a result of prolonged intravenous fluid administration with sufficient sodium but no dextrose.

Hypernatremia. Hypernatremia is an uncommon problem in the clinical setting. When hypernatremia occurs, it is usually related to water deficit as a result of prolonged intravenous fluid administration with sufficient sodium but no dextrose. This condition is readily reversible by a water solution supplemented with dextrose (Eggleston, 1985).

Potassium Abnormalities

Potassium is the major cation in the intracellular fluid (ICF), therefore it has a narrow normal range (3–5 mEq/L) outside the cells. The potassium concentration in the ECF may be higher than normal (hyperkalemia) or lower than normal (hypokalemia). The clinical signs of potassium abnormality are outlined in Table 12-16.

Potassium deficiency may be caused by excessive K$^+$ loss (trauma, severe infection, vomiting, use of diuretics) or inadequate K$^+$ intake (massive or prolonged intravenous fluid infusion without supplemental potassium).

Hypokalemia. Hypokalemia is a more common form of potassium (K$^+$) abnormality than hyperkalemia. Potassium deficiency may be caused by excessive K$^+$ loss (e.g., trauma, severe infection, vomiting, use of diuretics) or inadequate K$^+$ intake (e.g., massive or prolonged intravenous fluid infusion without supplemental potassium). Normal breakdown of body tissue produces some potassium as a by-product, but hypokalemia may still occur if excretion exceeds production.

Deficiency of serum potassium may be corrected by oral intake or slow intravenous infusion of potassium chloride. Potassium chloride is used because hypochloremia (low chloride) usually coexists with hypokalemia and the chloride ions must be replaced at the same time.

TABLE 12-16 Clinical Signs of Potassium Abnormality

System	Hypokalemia	Hyperkalemia
Neuromuscular	Decreased muscle functions	Increased neuromuscular conduction
Cardiac	Flattened T wave and depressed ST segment on ECG	Elevated T wave and depressed ST segment on ECG (mild)
	Arrhythmias	Cardiac arrest (severe)
Gastrointestinal	Decreased bowel activity Diminished or absent bowel sounds	Increased bowel activity Diarrhea

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✓ Oral intake of potassium replacement is safer. If an intravenous route is used, precautions must be followed to ensure patient safety.

Oral intake of potassium replacement is safer. If an intravenous route is used, there are four precautions that must be followed to ensure patient safety (Eggleston, 1985): (1) Consider replacement only if the urine output is at least 40 to 50 mL/hour; (2) Never use KCl undiluted as it can cause arrhythmias and cardiac arrest; (3) Do not give more than 40 mEq of potassium in any one hour or more than 200 mEq in 24 hours; and (4) Concentration of potassium in the intravenous drip should not be higher than 40 mEq/L.

✓ Hyperkalemia is usually caused by renal failure.

Hyperkalemia. Hyperkalemia is an uncommon condition in the clinical setting, but when hyperkalemia occurs it is usually due to renal failure. Decrease in urine output (less than 200 to 300 mL/day) secondary to renal failure leads to retention of potassium ions. Therefore, the primary treatment for this form of hyperkalemia is to improve kidney function.

In acute hyperkalemia, intravenous (IV) calcium chloride or calcium gluconate may aid in antagonizing the cardiac toxicity provided that the patient is not receiving digitalis therapy. Cellular uptake of potassium (from extracelluar compartment) may be increased by using sodium bicarbonate IV, regular insulin, and glucose IV. Beta-adrenergic (e.g., albuterol) shows various results. Elimination of total body potassium may be enhanced by using sodium polystyrene sulfonate (Kayexalate) orally (PO)/rectally (PR), furosemide (with normal renal function). Emergency hemodialysis is the treatment for life threatening hyperkalemia (Verive et al., 2010).

NUTRITION

Nutritional intake should be adjusted according to a patient's requirements. Inadequate intake may lead to impaired respiratory function due to reduction in the efficiency of respiratory muscles. Excessive intake may increase the patient's work of breathing due to the increased metabolic rate and carbon dioxide production.

Undernutrition

✓ Inadequate nutritional support can lead to fatigue of respiratory muscles.

Proper nutritional support is a therapeutic necessity for patients on a mechanical ventilator. Poor nutritional status may lead to rapid depletion of cellular stores of glycogen and protein in the diaphragm (Mlynarek et al., 1987). It also leads to fatigue of the major respiratory muscles in patients with or without lung diseases and contributes to impaired pulmonary function, hypercapnia, and inability to wean (Fiaccadori & Borghetti, 1991). Risk of infection becomes more likely when a patient is undernourished because of resultant decreased cell-mediated immunity. Interstitial and pulmonary edema may develop because of severe hypoalbuminemia in which the osmotic pressure is decreased and the fluid is shifted into the interstitial space (interstitial edema), and eventually into the alveoli (pulmonary edema). Other complications of undernutrition include poor wound healing and decreased surfactant production (Table 12-17) (Ideno et al., 1995).

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TABLE 12-17 Effects of Undernutrition

1.	Depletion of cellular stores of glycogen and protein
2.	Fatigue of respiratory muscles
3.	Impaired pulmonary function
4.	Decreased cell-mediated immunity
5.	Interstitial or pulmonary edema
6.	Poor wound healing
7.	Decreased surfactant production

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Overfeeding

✓ High caloric enteric nutrition can cause a significant increase in oxygen consumption and carbon dioxide production. In turn, this can induce respiratory distress during weaning for patients with limited pulmonary reserve.

While undernutrition is undesirable for critically ill patients, overfeeding should be avoided. Excessive nutrition may significantly increase the work of breathing because of lipogenesis and increased carbon dioxide production. It may also lead to diminished surfactant production and fatty degeneration of the liver (Table 12-18) (Ideno et al., 1995).

High caloric enteric nutrition can cause a significant increase in oxygen consumption, carbon dioxide production, and respiratory quotient. In turn, this can induce respiratory distress during weaning for patients with a limited pulmonary reserve.

Problems with overfeeding may also be found in total parenteral nutrition (TPN) provided via the intravenous route. Respiratory acidosis during mechanical ventilation has been reported within hours after initiation of TPN (van der Berg et al., 1988).

Low-Carbohydrate High-Fat Diet

Each gram of hydrous dextrose (a form of glucose) produces 3.4 kcal. For the same amount of fat emulsion, it generates 9.1 kcal. The concentrated source of

TABLE 12-18 Effects of Overfeeding

1.	Increased oxygen consumption
2.	Increased carbon dioxide production
3.	Increased work of breathing
4.	Decreased surfactant production
5.	Interstitial or pulmonary edema
6.	Fatty degeneration of liver

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energy in fat emulsion is preferred for fluid-restricted patients. A fat-based diet also reduces carbon dioxide production and ventilatory requirements (Mlynarek et al., 1987).

For this reason, an increase in fat kilocalories with a concurrent decrease in carbohydrate (dextrose) intake has been done to maximize energy intake and to minimize oxygen utilization and carbon dioxide production. The fat-based diet should contain at least 40% total fat kilocalories and it should be based on the patient's clinical status, because a metabolically stressed patient may become immunosuppressed because of insufficient fat in the diet (Ideno et al., 1995).

✔ A low-carbohydrate high-fat diet may maximize energy intake and minimize oxygen utilization and carbon dioxide production.

In one study, a high-calorie diet consisting of 28% carbohydrate, 55% fat, and balanced protein resulted in significantly lower CO_2 production and arterial PCO_2 in COPD patients with hypercapnia. Furthermore, two important lung function measurements (forced vital capacity and forced expiratory volume in 1 sec) improved by 22% over baseline values with this low-carbohydrate, high-fat diet (Angelillo et al., 1985).

Total Caloric Requirements

Energy requirements for the critically ill patient are commonly done by using the Harris-Benedict equation (Roza et al., 1984). This equation can be used to estimate a patient's resting energy expenditure (REE) and total energy expenditure (TEE). For an accurate measurement of a patient's energy requirement (REE and TEE), metabolic testing should be done.

REE is the minimum energy requirement for basic metabolic needs. TEE is the energy requirement based on a patient's disease state in which the metabolic rate is higher than normal. TEE is the product of REE and the activity/stress factors (TEE = REE × Activity × Stress Factors). These factors are used to make allowances for hypermetabolic or hypercatabolic conditions such as activity, trauma, infection, and burns. For ventilator-dependent patients, the TEE is calculated by multiplying the REE by factors ranging from 1.2 to 2.1 as shown in Table 12-19 (Askanazi et al., 1982; Roza et al., 1984).

Phosphate Supplement

The incidence of phosphate deficiency or hypophosphatemia is high in certain subgroups of patients. It occurs in about 30% of patients admitted to the ICU, 65% to 80% of patients with sepsis, 75% of patients with major trauma, and 21.5% of patients with COPD (Brunelli et al., 2007). In addition to the total caloric requirement, a patient's nutritional program should maintain a balanced serum phosphate level. Insufficient phosphate in a patient's diet may cause hypophosphatemia, a condition where the serum phosphate level is less than 1 mg/dL. Hypophosphatemia decreases tissue adenosine triphosphate (ATP) level, and in severe form it may cause the patient to experience confusion, muscle weakness, congestive heart failure, and respiratory failure (Mlynarek et al., 1987).

✔ Hypophosphatemia (serum phosphate level <1 mg/dL) in severe form may cause the patient to experience confusion, muscle weakness, congestive heart failure, and respiratory failure.

TABLE 12-19 Calculation of Daily REE and TEE in Kilocalories

REE for men in kcal/day $= 66 + 13.7\,W + 5\,H - 6.8\,A$	
REE for women in kcal/day $= 655 + 9.6\,W + 1.85\,H - 4.7\,A$	
W = weight in kg; H = height in cm; A = age in years	
TEE for men in kcal/day = REE × Activity Factor × Stress Factor	
TEE for women in kcal/day = REE × Activity Factor × Stress Factor	
W = Weight in kg; H = Height in cm; A = Age in year	
Activity factor	
Confined to bed	×1.2
Out of bed	×1.3
Stress factor	
Minor operation	×1.20
Skeletal trauma	×1.35
Major sepsis	×1.60
Severe thermal burn	×2.10

ADJUNCTIVE MANAGEMENT STRATEGIES

On some occasions, the basic management strategies may not be able to maintain proper ventilation and oxygenation. In other conditions such as acute lung injury (ALI) and adult respiratory distress syndrome (ARDS), the ventilator settings may result in volume and pressure that may be inappropriate and detrimental to the patient. Under these conditions, other management strategies should be considered. They include the use of low tidal volume, prone positioning, and trachea gas insufflation.

Low Tidal Volume

Traditional tidal volume settings use 10 to 15 mL/kg of body weight and this range is sometimes necessary to achieve normal ventilation. In one study, 48% of the critical care practitioners reported using volumes in the range of 10–15 mL/kg and 45% reported using 5–9 mL/kg (Thompson et al., 2001). In patients with ALI or ARDS, the inspiratory pressures (i.e., peak inspiratory and plateau) are often elevated due to an increased airflow resistance or/and a decreased lung compliance. The high inspiratory pressures lead to excessive distention of the normal aerated lung and may increase the incidence of **barotrauma (volutrauma)**. Therefore, the traditional approach in the selection of tidal volume may exacerbate or perpetuate lung injury in patients with ALI or ARDS and increase the risk of mortality and nonpulmonary organ and system failure (Petrucci et al., 2004; The Acute Respiratory Distress Syndrome Network, 2000).

barotrauma (volutrauma): Air leak into the pleural space caused by excessive pressure or volume in the lung parenchyma.

✓ The tidal volume selected for patients with ALI or ARDS should result in a plateau pressure of <35 cm H_2O.

Volume Selection. In volume-targeted ventilation for patients with ALI or ARDS, the tidal volumes selected should result in a plateau pressure of <35 cm H_2O (Thompson et al., 2001). Plateau pressure is used as a target pressure because it reflects the condition of the lung parenchyma. For the reason of lung protection, the lowest tidal volume that meets the patient's minimal oxygenation and ventilation requirements should be used.

Complications. Use of low tidal volume ventilation should be done with care as it may lead to complications such as acute hypercapnia, increased work of breathing, dyspnea, severe acidosis, and atelectasis (Kallet et al., 2001).

Prone Positioning

prone positioning (PP): Placement of the patient in a face-down position in a bed.

✓ PP has been used to improve ventilation, oxygenation, and pulmonary perfusion in patients with acute respiratory failure and ARDS.

Prone positioning (PP) has been used as a "stop-gap" strategy to improve the ventilation, oxygenation, and pulmonary perfusion status of patients with acute respiratory failure and ARDS. Following PP, there is a rapid increase in oxygenation measurements (e.g., SpO_2, PaO_2, SaO_2) and improvement in lung compliance (Relvas et al., 2003). The oxygen requirement, intrapulmonary shunting, and inspiratory pressures are reduced as well (Breiburg, 2000; Fletcher et al., 2003). Table 12-20 outlines the physiologic goals of PP.

While PP improves these pulmonary parameters rapidly, the improvements do not persist after the patient is returned to the original supine position. In addition, prone positioning does not increase the survival rate of patients with acute

TABLE 12-20 Physiologic Goals of Prone Positioning
To improve oxygenation (e.g., SpO_2, PaO_2, SaO_2)
To improve respiratory mechanics (e.g., compliance, work of breathing)
To enhance pleural pressure gradient, alveolar inflation, and gas distribution
To reduce inspiratory pressures (e.g., peak and plateau)
To reduce atelectasis and intrapulmonary shunting
To facilitate removal of secretions
To reduce ventilator-related lung injury

(Data from Breiburg, 2000; Fletcher et al., 2003; Pelosi et al., 2002; Relvas et al., 2003.)

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✓ PP improves oxygenation parameters rapidly but it does not increase the survival rate of patients with acute respiratory failure or ARDS.

respiratory failure or ARDS when compared to similar patients who are in the standard supine position (Gattinoni et al., 2001; Meade, 2002; Rialp et al., 2002).

Indications and Contraindications. The primary indication for PP is ARDS with increasing oxygen index (OI) of >30% while supine and during mechanical ventilation. OI requires measurement of the mean airway pressure (mPaw), F_IO_2, and PaO_2. See equation below and Appendix 1 for example to calculate the OI.

$$\text{OI} = \frac{(\text{mPaw} \times F_IO_2)}{PaO_2}$$

Contraindications for PP include increased intracranial pressure, hemodynamic instability, unstable spinal cord injury, recent abdominal or thoracic surgery, flail chest, and inability to tolerate PP.

✓ After 1 hour of PP, an improvement of the OI by >20% of baseline value suggests beneficial response.

Procedure. If no contraindication for PP exists, the patient is turned to a prone position for at least 1 hour (stabilization period). After 1 hour, the PaO_2/F_IO_2 ratio and the mPaw are measured. An improvement of the OI by ≥20% of baseline value suggests beneficial response to PP.

For optimal improvement in oxygenation and more stable improvement in the OI, pediatric patients should remain in the PP for a period longer than 12 hours. The procedure for PP (preparing the patient, placing the patient in PP and SP) has been fully described by Relvas et al. in 2003. For adult patients, the duration of PP should be 6 hours or more depending on patient response and tolerance (Gattinoni et al., 2001; Meade, 2002).

Complications. Complications of PP include accidental extubation, hemodynamic instability, pressure wounds or ulcers, residual obstructive and restrictive lung defects, and **brachial plexopathy** (Curley et al., 2000; Goettler et al., 2002; Neff et al., 2003; Relvas et al., 2003).

brachial plexopathy: Decreased movement or sensation in the arm and shoulder.

Tracheal Gas Insufflation

tracheal gas insufflation (TGI): Use of a small catheter to provide a continuous or phasic gas flow directly into the trachea during mechanical ventilation.

Tracheal gas insufflation (TGI) is a technique that uses a small catheter to provide a continuous or phasic gas flow directly into the endotracheal tube during mechanical ventilation. Slusky and Menon described in 1987 the use of a constant-flow device in conjunction with ventilation. Over the years, innovations have been made on similar techniques.

✓ TGI introduces 5 to 20 L/min of oxygen or air into the endotracheal (ET) tube during mechanical ventilation.

Procedure. TGI introduces 5 to 20 L/min of oxygen or air into the endotracheal (ET) tube during mechanical ventilation. This flow is in addition to the flow provided by the ventilator. The flow provided by the TGI is regulated by a controller and is directed through a small catheter to the distal end of the ET tube. The gas exits the ET tube and arrives just above the carina (Valley Inspired Products, Burnsville, MN).

The insufflation may be continuous or phasic. In continuous-flow TGI, the gas flow goes into the airway during inspiration and expiration. Some undesirable effects of continuous TGI include drying of secretions, mucosal tissue damage, increased tidal volume delivery, development of auto-PEEP, and increased effort to trigger the ventilator. In phasic TGI, the gas flow goes into the airway during the last half of

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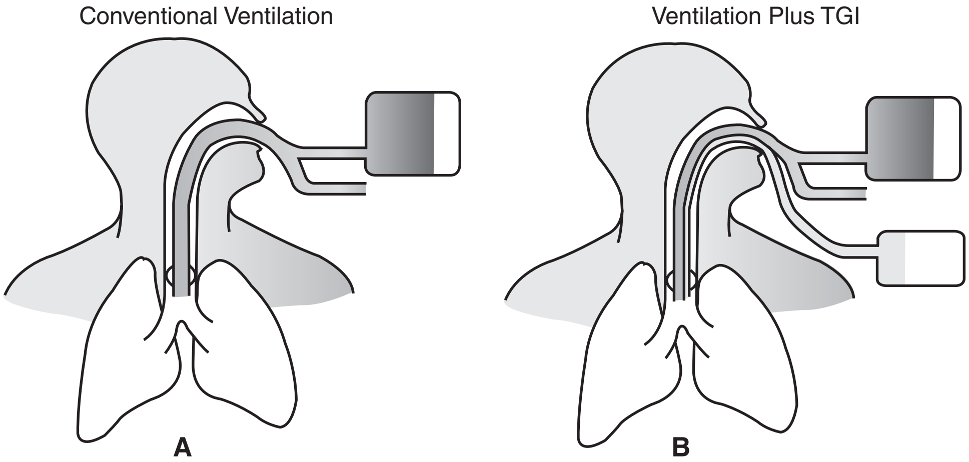

Courtesy of Valley Inspired Products, Burnsville, MN. Used with permission.

FIGURE 12-8 Conventional ventilation and tracheal gas insufflation. (A) In conventional ventilation, expired gas (~4% CO_2) remains in the endotracheal tube and goes back into the lung on the next breath. (B) With tracheal gas insufflation, the expired gas is flushed from the endotracheal tube with fresh gas (0% CO_2). This fresh gas goes into the lungs on the next breath.

the expiratory phase. This phasic timing helps to flush out the ET tube with fresh gas (0% carbon dioxide) during expiration and fills the ET tube with fresh gas for the next inspiration (Figure 12-8).

✓ TGI reduces the instrumental deadspace, improves carbon dioxide clearance, and lowers the ventilation pressure and tidal volume requirements.

Potential Uses. During mechanical ventilation of newborns, TGI reduces the instrumental deadspace, improves carbon dioxide clearance, reduces carbon dioxide rebreathing, and lowers the ventilation pressure and tidal volume requirements. The $PaCO_2$ may be reduced with no change in minute ventilation, or the $PaCO_2$ may be maintained at the same level with 10% to 20% reduction in minute ventilation. These effects of TGI have the potential to decrease the likelihood of secondary lung injury and chronic lung disease in newborns (Davies et al., 2002; Epstein, 2002; Kalous et al., 2003; Liu et al., 2004; Virag, 2011). TGI has also been used successfully to reduce the respiratory demand during weaning from mechanical ventilation (Hoffman et al., 2003).

TGI is a modality that has the potential to improve the management of patients with acute respiratory failure. Lack of a simple and reliable patient interface for TGI is one of the problems in the approval process by the FDA (Virag, 2011). Additional research studies and more clinical trials are necessary to make TGI an FDA-approved device for the general patient population.

SUMMARY

This chapter outlines the essential strategies that are useful in the management of common ventilator-related issues. These strategies are straightforward and can be followed by using a logical deduction process. Careful observation of the patient and ventilator must be done in order to identify the problem. Once the problem is identified, appropriate steps may be taken. It is vital to remember that no changes to the ventilator settings should be made unless the reasons for doing so are justifiable based on clinical data and patient presentations.

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Self-Assessment Questions

1. Strategies that are useful to improve ventilation include all of the following *except*:

    A. increase mechanical deadspace.
    B. increase ventilator tidal volume.
    C. increase ventilator frequency.
    D. increase pressure support for spontaneously breathing patients.

2. Dr. McFarland asks the therapist to adjust the ventilator in order to improve the oxygenation status of the patient with normal V/Q status. Which of the following ventilator adjustments would have the most direct effect on oxygenation?

    A. decrease tidal volume (V_T)
    B. decrease pressure support ventilation (PSV)
    C. increase mandatory frequency (f)
    D. increase oxygen concentration (F_IO_2)

3. An endotracheal tube is sometimes shortened because a shorter ET tube:

    A. increases the F_IO_2.
    B. increases the mechanical deadspace volume.
    C. facilitates airway management and secretions removal.
    D. reduces the arterial pH.

4. The primary purpose of permissive hypercapnia is to reduce the patient's ________ during mechanical ventilation.

    A. tidal volume
    B. pH
    C. pulmonary pressures
    D. frequency

5. Permissive hypercapnia is a technique in which the mechanical ________ is reduced. This change is done intentionally to increase a patient's ________.

    A. peak airway pressure, pH
    B. peak airway pressure, PaO_2
    C. tidal volume, pH
    D. tidal volume, $PaCO_2$

6. CPAP and PEEP may be used to reduce or correct refractory hypoxemia caused by:

    A. deadspace ventilation.
    B. V/Q mismatch.
    C. intrapulmonary shunting.
    D. diffusion defect.

7. The PaO_2 of a spontaneously breathing patient has been deteriorating while on 60% of oxygen via a partial-rebreathing mask. The physician asks the therapist to suggest the best solution for this problem. The therapist should recommend the following procedures in the order provided:

   A. CPAP, mechanical ventilation with PEEP, inverse ratio ventilation.
   B. inverse ratio ventilation, mechanical ventilation, PEEP.
   C. mechanical ventilation, CPAP, inverse ratio ventilation with PEEP.
   D. mechanical ventilation, inverse ratio ventilation, PEEP.

8. Compensated respiratory acidosis and compensated metabolic alkalosis have similar blood gas characteristics: normal pH, high $PaCO_2$, and high HCO_3^-. One useful clue to differentiate these two conditions is that in compensated:

   A. respiratory acidosis, the pH is on the acidotic side of normal range.
   B. metabolic alkalosis, the pH is on the acidotic side of normal range.
   C. respiratory and metabolic acidosis, both pH are on the alkalotic side of normal range.
   D. respiratory and metabolic alkalosis, both pH are on the acidotic side of normal range.

9. A patient's low pressure alarm is triggered persistently. The likely causes of this condition include all of the following *except*:

   A. disconnection of ventilator circuit.
   B. kinking of endotracheal tube.
   C. power failure.
   D. leakage of endotracheal tube cuff.

10. During patient rounds in the ICU, the high pressure alarm of a ventilator is triggered. This condition is likely caused by:

    A. disconnection of ventilator circuit.
    B. low pressure limit set too high.
    C. loose ventilator humidifier fitting.
    D. patient coughing.

11. Analysis of sputum samples by the culture and sensitivity method is ________. It provides information on the type of ________ that the microbes are sensitive to.

    A. quick, sterilizing agents
    B. quick, antibiotics
    C. time-consuming, sterilizing agents
    D. time-consuming, antibiotics

12. The urine output of a patient is about 15 mL/hour. This volume of urine output is ________ than normal and it implies that there is too ________ fluid in the extracellular fluid compartment.

    A. higher, much   B. higher, little
    C. lower, much   D. lower, little

13. The following electrolyte values are collected from a patient with severe sepsis who has been on a mechanical ventilator for 2 weeks. Which of the following electrolytes is out of normal range?

Electrolyte	Value (mEq/L)
A. Na^+	138
B. K^+	1.5
C. Cl^-	105
D. HCO_3^-	25

14. In replacing fluids to a volume-depleted patient, it is not safe to administer fluids that have no sodium because ________ movement of sodium-free fluid into the brain and kidney cells may cause ________ of these organs.

A. rapid, swelling
B. rapid, dehydration
C. slow, swelling
D. slow, dehydration

15. Decreased muscle function, flattened T wave and depressed ST segment on the electrocardiogram, and diminished bowel sounds are some signs of:

A. hyperkalemia.
B. hypokalemia.
C. hypernatremia.
D. hyponatremia.

16. Proper nutrition is essential to patients receiving mechanical ventilation because undernutrition can cause:

A. increased surfactant production.
B. improved pulmonary function.
C. increased metabolic rate.
D. fatigue of respiratory muscles.

17. A diet consisting of low carbohydrate and high fat is more suitable for ventilator patients because ________ generates more calories per gram and produces less ________.

A. fat, CO_2
B. fat, O_2
C. carbohydrate, CO_2
D. carbohydrate, O_2

18. The total energy expenditure (TEE) is ________ than the resting energy expenditure (REE) because TEE ________ accounts for patient factors such as activity, trauma, and infection.

A. higher, does
B. higher, does not
C. lower, does
D. lower, does not

19. Mr. Howe, a 70-kg patient with ARDS, is being mechanically ventilated at a SIMV frequency of 12/min and tidal volume of 600 mL. Over a period of 8 hours, the peak inspiratory and plateau pressures have increased to 66 and 45 mm Hg, respectively. The attending physician asks a therapist to suggest changes to minimize the effects of rising airway pressures. The therapist should recommend initiation of:

A. ventilation with low frequency.
B. ventilation with low tidal volume.
C. tracheal gas insufflation.
D. prone positioning.

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Answers to Self-Assessment Questions

1. A.
2. D.
3. C.
4. C.
5. D.
6. C.
7. A.
8. A.
9. B.
10. D.
11. D.
12. D.
13. B.
14. A.
15. B.
16. D.
17. A.
18. A.
19. B.

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Chapter 13

Pharmacotherapy for Mechanical Ventilation

Sandra Gaviola
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Outline

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Key Terms

acetylcholine
antiemetic
anxiolysis
barbiturates
benzodiazepines
cathartic agents
chronotropic
corticosteroids
depolarizing agents
dexmedetomidine (Precedex)
gamma-aminobutyric acid (GABA)
haloperidol (Haldol)
inotropic
nitric oxide
nondepolarizing agents
opioid analgesics
parasympatholytic bronchodilators
propofol (Diprivan)
Ramsay Scale
sympathomimetic bronchodilators
xanthine bronchodilators

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Provide the mechanism of action, adverse effects, and examples of: adrenergic, anticholinergic, xanthine bronchodilators, and anti-inflammatory agents.
- Differentiate depolarizing and nondepolarizing neuromuscular blocking agents and provide the mechanism of action and examples of these agents.
- Provide the mechanism of action, adverse effects, and examples of sedatives and antianxiety agents.
- Provide the mechanism of action, adverse effects, and examples of opioid analgesics.
- Provide the mechanism of action, adverse effects, and examples of barbiturates.
- List the clinical application of propofol, haloperidol, dexmedetomidine, and nitric oxide.

INTRODUCTION

Two primary purposes of drug therapy for patients using mechanical ventilation are that they (1) provide patient comfort and (2) facilitate airway management and mechanical ventilation. For example, some drugs are used to facilitate intubation (neuromuscular blockers and sedatives) and reduce airflow resistance (bronchodilators), while others are necessary to manage pain (opioids) and induce sedation (sedatives). Proper use of drug therapy is necessary to achieve desired outcomes. A clear understanding of these drugs is also essential to avoid misuse, complications, and prolonged mechanical ventilation.

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DRUGS FOR IMPROVING VENTILATION

Airway narrowing is a common complication in patients receiving mechanical ventilation. Increasing peak inspiratory pressure, wheezing, hypoxemia, and agitation are some clinical signs that indicate the presence of airway distress.

For the ventilator patient, airway distress may be caused by (1) preexisting airway disease (chronic bronchitis, asthma), (2) drug-induced bronchospasm, (3) accumulated secretions, and (4) mechanical irritation. Whatever the cause, the distress must be recognized and corrected quickly to prevent hypoxemia and further deterioration.

Along with bronchial hygiene procedures, bronchodilators and corticosteroids play a critical role in achieving optimal airway patency and constitute the majority of nebulized drugs used in respiratory care.

Autonomic Nervous System Agents

The smooth muscles of the airway are under autonomic (involuntary) nervous control. Patients do not have control over the patency of the affected airways. When airways constrict due to different mechanisms, bronchodilators are commonly used to provide airway dilation and patient relief. The following sections discuss the bronchodilators that exert their effects on two major pathways (i.e., sympathetic and parasympathetic) under the autonomic nervous system (Figure 13-1).

Sympathetic and Parasympathetic Branches. The autonomic nervous system (ANS) comprises motor neurons that innervate tissues under involuntary control. Among important functions regulated by the ANS are respiration, heart rate, blood pressure, perspiration, and glandular secretions. The sympathetic and parasympathetic fibers are the basic subdivisions of the ANS (the enteric pathway is a third subdivision not of direct interest to this discussion), and, for the most part, they elicit responses in an opposing manner (at the effector sites). For example, stimulation of the sympathetic branch results in bronchodilation, whereas stimulation of the parasympathetic branch causes bronchoconstriction (Tortora et al., 2002).

For example, the interaction of the sympathetic and parasympathetic branches can be represented by two teams in a very well-balanced tug-of-war match. The goal is to keep the flag tied to the middle of the rope from crossing the line on either team's side. Both teams always pull to keep tension on the rope (basal tone) but occasionally the sympathetic team gives an extra surge of effort to pull the flag in its direction, and the parasympathetic team quickly responds by increasing its effort until the other team tires and the flag is once more pulled back toward the middle ground. Bronchodilators produce appropriate changes in the sympathetic or parasympathetic branch.

Adrenergic and Cholinergic Responses. The sympathetic pathway terminal axon uses the neurotransmitter substance epinephrine (adrenaline)/norepinephrine, from which the term *adrenergic response* is derived.

The neurotransmitter substance released at the terminal axon of the parasympathetic fiber is acetylcholine (ACh) and it elicits a cholinergic response.

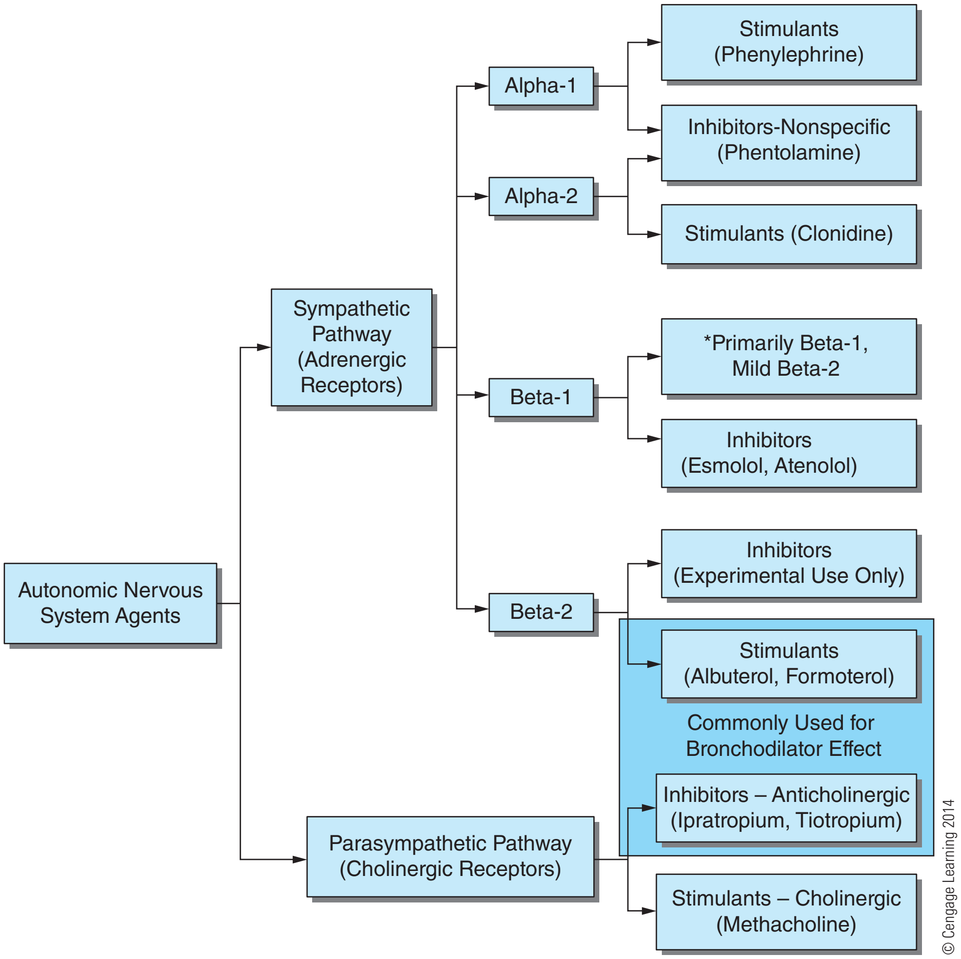
Autonomic Nervous System Agents
Sympathetic Pathway (Adrenergic Receptors)
Parasympathetic Pathway (Cholinergic Receptors)
Alpha-1
Alpha-2
Beta-1
Beta-2
Stimulants (Phenylephrine)
Inhibitors-Nonspecific (Phentolamine)
Stimulants (Clonidine)
*Primarily Beta-1, Mild Beta-2
Inhibitors (Esmolol, Atenolol)
Inhibitors (Experimental Use Only)
Stimulants (Albuterol, Formoterol)
Commonly Used for Bronchodilator Effect
Inhibitors – Anticholinergic (Ipratropium, Tiotropium)
Stimulants – Cholinergic (Methacholine)
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FIGURE 13-1 Examples of drugs that exert their effects on the sympathetic and parasympathetic pathways under the autonomic nervous system.

sympathomimetic bronchodilators: Adrenergic agonists. Drugs that dilate the airways by stimulating the beta-2 receptors of the sympathetic nervous system. Examples are epinephrine (Adrenaline) and albuterol (Ventolin, Proventil).

parasympatholytic bronchodilators: Anticholinergic bronchodilators. Drugs that dilate the airways by inhibiting the parasympathetic branch of the autonomic nervous system. Examples are atropine and ipratropium bromide (Atrovent).

Bronchodilation can be achieved by eliciting an adrenergic response (sympathomimetic action) or by interfering with the cholinergic response (parasympatholytic action). Many bronchodilators use the sympathomimetic action (**sympathomimetic bronchodilators**) while other bronchodilators take advantage of the parasympatholytic action (**parasympatholytic bronchodilators**). Table 13-1 shows these two pathways to achieve bronchodilation.

Adrenergic Bronchodilators (Sympathomimetics)

Adrenergic bronchodilators are agents that stimulate the adrenergic receptors via the sympathetic nerve fibers of the autonomic nervous system.

Mechanism of Action. Sympathetic adrenergic receptors are distributed throughout the body. They are identified and classified according to their response to specific neurotransmitter substances. Alpha 1, alpha 2, beta 1, and beta 2 are the types that have been identified. Some important effects associated with these receptors are shown in Table 13-2.

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TABLE 13-1 Two Autonomic Nervous System (ANS) Pathways for Bronchodilation

Sympathetic Pathway	Parasympathetic Pathway
Stimulation of adrenergic response	Interference of cholinergic response
↓	↓
↑↑ Sympathomimetic activity	↑ Parasympatholytic activity
(Stimulation of sympathetic activity)	(Inhibition of parasympathetic activity)
↓	↓
Bronchodilation	Relative increase of sympathetic activity
	↓
	Bronchodilation

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In the lungs, β-2 receptors are found in the smaller airways, alveolar walls, and submucosal glands. The primary goal of beta-2 adrenergic drugs is to combine with these receptors to initiate bronchodilation.

Adrenergic bronchodilators are classified as catecholamines or catecholamine derivatives.

Catecholamines. Catecholamines such as norepinephrine, epinephrine, and isoproterenol share the following characteristics: (1) rapid onset, (2) rapid degradation by catechol-O-methyltransferase (COMT) in the liver, kidney, and throughout the body, and the intraneuronal enzyme monoamine oxidase (MAO), (3) ineffective when taken enterally, and (4) nonspecific receptor binding.

Catecholamine derivatives. Catecholamine derivatives such as metaproterenol, terbutaline, albuterol, and pirbuterol have a more complex chemical structure than

TABLE 13-2 Major Effects of Alpha and Beta Receptors

Receptor	Major Effects
Alpha-1 (α-1)	Vasoconstriction Constriction of pupils
Alpha-2 (α-2)	Decreased gastrointestinal activity
Beta-1 (β-1)	Positive **inotropic** effect (↑ muscular contractility) Positive **chronotropic** effect (↑ heart rate)
Beta-2 (β-2)	Bronchodilation Peripheral vasodilation Decreased gastrointestinal activity

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inotropic: Affecting the contraction.

chronotropic: Affecting the rate.

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catecholamines. The modification in the chemical structure results in more specific beta-2 receptor binding and delayed degradation by COMT and MAO. As a result, catecholamine derivatives (noncatecholamines) offer less cardiac adverse effects and prolonged bronchodilation than the catecholamines. Additionally, resistance to COMT degradation makes these agents suitable for enteral administration—although, beta-2 specificity may be lost via this route (Gardenhire, 2007). Long-acting beta adrenergic medications such as aformoterol tartrate (Brovana) have the benefit of less frequent dosing, but the question of increased risk of death when used as monotherapy with patients who have asthma remains unresolved (Hinkle, 2011).

Table 13-3 shows the relative receptor actions, dosage, and frequency of use of some common sympathomimetics.

Adverse Effects. The adverse effects of adrenergic bronchodilators include tachycardia, palpitations, skeletal muscle tremors, and nervousness. The degree of adverse effects depends on the mode of administration, dosage, frequency of administration, presence of preexisting cardiac disease, and the specific adrenergic agent used. In most cases the benefits of bronchodilation will outweigh the potential adverse effects.

TABLE 13-3 Adrenergic Bronchodilators

Catecholamines	**Action Receptor**	**Inhalation**	**Dosage**	**Frequency**
Epinephrine (adrenaline)	α > β-1 > β-2	Neb (1%)	0.25 to 0.5 mL	QID
Racemic Epinephrine (Micro-Nefrin, Vaponefrin, Asmanefrin)	α > β-1 > β-2	Neb (2.25%)	0.25 to 0.5 mL	QID
Isoproterenol (Isuprel)	β-1 < β-2	Neb (0.5%)	0.25 to 0.5 mL	QID
Catecholamine Derivatives				
Bitolterol (prodrug) (Tornalate)	β-1 < β-2	MDI	2 puffs	Q 4 to 6°
Metaproterenol (Alupent, Metaprel)	β-1 < β-2	Neb MDI	(5%) 0.3 mL 2 to 3 puffs	TID/QID Q 4°
Albuterol (Proventil, Ventolin)	β-1 < β-2	Neb (0.5%) MDI, DPI	0.5 mL 2 puffs	TID/QID TID/QID
Levalbuterol (Xopenex)	β-1 < β-2	Neb, MDI	0.63 to 1.25 mg	TID/QID
Terbutaline (Brethaire, Brethine, Bricanyl)	β-1 < β-2 DPI	MDI 1 puff	2 puffs QID	QID
Pirbuterol (Maxair)	β-1 < β-2	MDI	1 to 2 puffs	Q 4 to 6°
Salmeterol Xinafoate (Serevent)	β-1 < β-2	MDI, DPI	2 puffs	BID
Arformoterol Tartrate (Brovana)	β-1 < β-2	Neb	15 mcg	BID

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Special Considerations. The primary purpose of beta agonists is bronchodilation. For patients with inflammatory airway disease (i.e., asthma and chronic bronchitis), intermittent use of beta agonists may not be sufficient. Deaths have been reported in asthmatics following regular and prolonged use of these agents. Uncontrolled inflammation and mucosal edema are likely the cause of this adverse outcome (Witek, 1994).

Desensitization of receptor sites has been documented with regular use of beta agonists. Two mechanisms appear to be responsible: (1) the loss of receptor from the surface of cells and (2) the failure of the cell to function (Cottrell et al., 1995). The practitioner may wish to combine beta agonists with other categories of drugs to achieve bronchodilation. For example, a combination of corticosteroid and beta agonist may potentiate their individual effects and produce bronchodilation.

However, paradoxical bronchospasm may occur with combined use of adrenergic agonists and corticosteroids. Its occurrence is rare, but the onset can be sudden. For this reason, practitioners should be aware of the hazard.

acetylcholine: An ester that plays a role in the transmission of nerve impulses at synapses and neuromuscular junctions. It is metabolized by an enzyme, cholinesterase. Too much or too little of acetylcholine at the motor endplates may lead to muscle blockade.

✓ Atropine is an anticholinergic agent used as a secondary bronchodilator. It is also used for symptomatic bradycardia and prophylactic drying of secretions before surgery.

Anticholinergic Bronchodilators (Parasympatholytics)

Anticholinergic bronchodilators are agents that impede the impulses of the cholinergic, especially the parasympathetic nerve fibers of the autonomic nervous system.

Mechanism of Action. Parasympathetic receptors (muscarinic and nicotinic) are found throughout the body. They are classified according to whether they respond to muscarine or nicotine. In the lungs, muscarinic receptors are found in submucosal glands, mast cells, and smooth muscles of the larger airways.

The combination of **acetylcholine** with muscarinic receptors results in increased bronchial tone and increased secretion from mucosal glands. Anticholinergic agents (atropine and atropine derivatives) block these physiologic responses and may be useful for the reversal of vagally mediated bronchospasm. Table 13-4 shows the usage of four common anticholinergic bronchodilators.

TABLE 13-4 Anticholinergic Bronchodilators

Drug	Inhalation	Dosage	Frequency
Tiotropium (Spiriva)	DPI (HandiHaler)	18 mcg	Once daily
Atropine	Neb	0.3 to 0.5 mL	Up to 4 times/day
Ipratropium bromide (Atrovent)	MDI Neb (0.025%)	1 to 2 puffs 1 to 2 mL	QID Q 4 to 6°
Glycopyrrolate (Robinul)	Neb (0.02%)	5 mL	Q 4 to 6°

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Adverse Effects. When inhaled, atropine is readily distributed throughout the body and may cause systemic effects such as tachycardia, nervousness, headache, and dried secretions. Atropine easily penetrates the blood-brain barrier and at higher levels may cause hallucination or mental confusion. Patients with atropine-induced hallucinations have been erroneously diagnosed with psychiatric disorders.

Other anticholinergic agents such as ipratropium bromide (Atrovent) and glycopyrrolate (Robinul, an atropine derivative) are not well absorbed systemically and, when inhaled, produce fewer adverse effects than those produced by atropine. Drying of secretions is an adverse effect of Atrovent, but it can be prevented by proper humidification or systemic hydration.

Clinical Considerations. Ipratropium bromide is not indicated for the initial treatment of acute episodes of bronchospasm where immediate response is required. These agents are commonly used in addition to the rapid-acting beta agonists. For example, ipratropium bromide is premixed with albuterol sulfate for MDI use (Combivent). The normal dosage is 2 puffs of this mixture QID.

Xanthine Bronchodilators

xanthine bronchodilators: Drugs that produce bronchodilation by inhibiting phosphodiesterase, an enzyme that inactivates cyclic 3′5′ AMP (a substance that promotes bronchodilation). Examples are oral theophylline (Theo-Dur, Slo-bid) and aminophylline, a water-soluble theophylline (Aminophyllin, Somophyllin).

The third class of bronchodilators, the **xanthines,** include the drugs theophylline and its salt form aminophylline. Theophylline is a stimulant found in tea leaves and is chemically related to other stimulants found in coffee and colas. Because of the structural similarity of caffeine and theophylline, they share effects such as tachycardia, central nervous stimulation (wakefulness), and diuresis (and potential for dysrhythmia). Caffeine is a xanthine that produces similar effects to theophylline but works through somewhat different mechanisms and therefore can have an additive effect (caffeine is included in aminophylline solution).

✔ Pulmonologists often refer to theophylline as "Dig- (digitalis)for the diaphragm," meaning that what digoxin does for heart contractility, theophylline does for diaphragm contracticility.

Xanthines are used for their relaxing effects on smooth muscles and ability to inhibit inflammation; however, be aware that in some individuals theophylline may aggravate bronchospasm because it can lower esophageal pressure, which may lead to reflux. Clinically, the xanthines are considered to be less effective in acute bronchospasm than the beta agonists and are more useful in the management of inflammation associated with asthma and COPD. For individuals with carbon dioxide retention, xanthines improve ventilation by heightening carbon dioxide sensitivity in the central nervous system and enhancing diaphragmatic contractility.

Water-soluble aminophylline is suitable for intravenous administration and may be indicated as an add-on therapy for acute bronchospasm (Cottrell et al., 1995). Table 13-5 lists the oral and intravenous xanthine bronchodilators. Theophylline's ability to enhance diaphragm contractility and respiratory drive are of primary concern for COPD patients, but there is renewed interest in the drug for asthmatic patients, particularly for its potential to reduce airway inflammation.

Mechanism of Action. Multiple theories on the mechanism of theophylline have been described. One generally accepted mechanism is that theophylline produces bronchodilation by inhibiting phosphodiesterase (PDE). Recall that PDE rapidly inactivates cyclic 3′5′ AMP, the substance that relaxes the airway smooth muscles

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TABLE 13-5 Xanthine Bronchodilators

Generic Name	Trade Names	Administration
Theophylline (100% anhydrous)	(Aerolate, Constant-T, Respbid, Slo-bid, Theo-Dur, Uniphyl)	Oral, IV
Aminophylline (78–86% theophylline, water soluble)	(Aminophyllin, Somophyllin)	Intravenous

✓ Inhibition of phosphodiesterase, acting as an adenosine antagonist, and increased catecholamine release are three proposed mechanisms of action of theophylline.

and inhibits mast cell histamine release. By inhibiting PDE, xanthine indirectly increases cyclic 3′5′ AMP levels. A second theory describes theophylline as an adenosine antagonist. Normally, the stimulation of adenosine receptors results in histamine release, thus a blocking effect provides anti-inflammatory benefits. And finally, theophylline has been shown to increase catecholamine release in some studies. The enhanced sympathomimetic-like adverse effects experienced with a nonautonomic drug such as theophylline seems to support this finding (Gardenhire, 2007).

Adverse Effects. Unlike the adrenergic and anticholinergic bronchodilators, xanthines are not useful via inhalation due to their lack of ability to penetrate the mucosal lining of the airways. Xanthines are available for systemic administration via the intravenous and oral routes and, thus, they produce widespread effects.

Some of the adverse effects (tachycardia and skeletal muscle and CNS stimulation) can occur even at therapeutic serum theophylline ranges of 5–15 mcg/mL. Routine monitoring of serum theophylline levels is one way to prevent these effects from intensifying to the degree of toxicity. However, the practitioner should be able to recognize the early warning signs of theophylline toxicity since a narrow margin between therapeutic and toxic levels exists. Patients with mild to moderate toxicity (20 to 30 mcg/mL) may experience tachypnea, palpitations, nausea, vomiting, headache, and agitation. Severe toxicity (> 40 mcg/mL) is marked by gastric bleeding, arrhythmias, and seizures (Wilkins et al., 2008; Witek, 1994).

✓ Nausea, vomiting, abdominal pain, diarrhea, and nervousness are some initial signs of theophylline toxicity.

✓ Adverse effects of theophylline can be minimized by keeping the serum theophylline level from 5 to 15 mcg/mL.

Clinical Considerations. Nausea, vomiting, abdominal pain, diarrhea, and nervousness are some initial signs of theophylline toxicity. These signs may not be apparent in or communicated by the sedated or paralyzed patient. If xanthines are used in a paralyzed patient, the serum theophylline level should be monitored closely to prevent inadvertent overdose. Toxic adverse effects of theophylline can be minimized when the serum theophylline level is maintained within its therapeutic range of 5–15 mcg/mL (NIH Publication, 1997).

Most of the theophylline is metabolized by the liver and excreted in the urine. Patients at risk for theophylline toxicity are those with heart failure or liver disease. Diminished liver perfusion (due to heart failure) or impaired liver function can reduce the metabolism and clearance rate of theophylline. The end result is excessive theophylline accumulation and toxicity.

On the other hand, patients at risk for inadequate theophylline are those who smoke. Smoking increases the level of hepatic enzyme and theophylline clearance.

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The end result for these individuals is higher maintenance theophylline dosages to maintain bronchodilation (Cottrell et al., 1995).

Anti-Inflammatory Agents (Corticosteroids)

In addition to the drugs that exert their bronchodilation effects using the pathways in the autonomic nervous system, **corticosteroids** are a different group of drugs that are useful in the management of bronchoconstriction and airflow obstruction due to airway inflammation. Corticosteroids are powerful, naturally occurring hormones that are released from the adrenal cortex. Their anti-inflammatory effects make them first-line drugs in the management of chronic asthma and other long-term airway inflammatory conditions. In the intensive care setting, they have been used with favorable results in status asthmaticus, acute exacerbation of COPD, inhalation airway injury, drug-induced pneumonitis, septic shock, ARDS, and spinal cord injury.

corticosteroids: Hormones that are released from the cortex of the adrenal gland. Their potent anti-inflammatory effects make corticosteroids useful in the treatment of asthma and chronic bronchitis. Corticosteroids are available for intravenous administration as well as inhalation. Examples of inhaled steroids are dexamethasone (Decadron, Respihaler), beclomethasone (Beclovent, Vanceril), flunisolide (AeroBid), and triamcinolone (Azmacort).

Corticosteroids have no bronchodilator effect and should not be given alone during an acute asthma attack. The effects of corticosteroids require an onset time of about 2 to 24 hours, further demonstrating their inappropriateness in acute situations.

Mechanism of Action. The general functions of corticosteroids include (1) carbohydrate metabolism, (2) immunosuppression, and (3) reduced inflammation. Their specific mechanism in reducing inflammation is complex, involving genetic changes to target cells of inflammation. The combination of the steroid with its receptor site (nucleus of target cell) results in altered cellular function.

Normally, an inflammatory response results in the release of histamine and bradykinin (among other mediators) from the mast cell, causing bronchoconstriction and increased capillary permeability. This leads to early-phase bronchoconstriction and late-phase submucosal edema and hyperactivity (Gardenhire, 2007). With altered cellular function, these and other mediators are blocked and inflammation is reduced.

✓ Corticosteroids return constricted airways to normal by blocking the inflammatory mediators.

Adverse Effects. Corticosteroids are available for oral, intravenous, and inhalation administration (Tables 13-6 and 13-7), and, as with most agents, the inhalation route is favored for its convenience, smaller dosage, and fewer adverse effects.

The most frequent adverse effects reported with a metered-dose inhaler (MDI) use are hoarseness and oral fungal infections (e.g., candidiasis). To prevent fungal

TABLE 13-6 Corticosteroids for Metered-Dose Inhaler (MDI) Use

Drug	MDI Dosage	Frequency
Beclomethasone (QVAR)	2 puffs	2 times/day
Flunisolide (AeroBid)	2 puffs	2 times/day
Triamcinolone (Azmacort)	2 puffs	3 to 4 times/day
Fluticasone propionate (Flovent)	2 puffs	2 times/day

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TABLE 13-7 Corticosteroids for Systemic Use

Drug	Route of Administration
Hydrocortisone	Oral, IV
Cortisone	Oral
Prednisolone	Oral
Dexamethasone	Oral, IV
Prednisone	Oral

superinfections, patients should be instructed to rinse and gargle the mouth after each treatment. MDI corticosteroids are rarely used during mechanical ventilation.

There are many serious adverse effects associated with systemic corticosteroid therapy. Abrupt withdrawal following long-term systemic therapy may cause serious complications from adrenal insufficiency. Recovery time varies depending on the dosage and duration of therapy. Another serious effect is an increased susceptibility to opportunistic infections.

✓ Corticosteroids may be used when other traditional bronchodilators have failed to relieve bronchospasm.

Clinical Considerations. Corticosteroids are not bronchodilators. Their use in the treatment of bronchospasm is limited to situations where patients have lost beta agonist responsiveness or when other bronchodilators have failed. Their primary role is in the management of long-term airway inflammation. Systemic corticosteroids should be used cautiously with patients receiving steroidal-based neuromuscular blocking agents (vecuronium bromide and pancuronium bromide) because of the potential of prolonged neuromuscular weakness (Kupfer et al., 1987).

DELIVERY OF MDI MEDICATIONS

✓ Delivery of MDI medications during mechanical ventilation can be enhanced by synchronization of actuation of MDI with onset of inspiratory flow, a tidal volume of 500 mL or more, a longer inspiratory time, and a slower inspiratory flow.

Many bronchodilators and steroids come in a metered-dose inhaler (MDI). With proper adaptor or fittings, MDI medications may be administered via the ventilator circuit without interruption to mechanical ventilation or airway pressure. The actuation of an MDI in a ventilator circuit must be synchronized with the onset of inspiratory flow. A slight delay (e.g., 1 to 1.5 sec) after the onset of inspiratory flow can significantly reduce the amount of drug delivery. A tidal volume of 500 mL or more, a longer inspiratory time, and a slower inspiratory flow improve drug delivery to the lower respiratory tract and lungs (Dhand, 2005).

MDI may be given with a right-angle adaptor or with a spacer. Studies have shown that an MDI with spacer is a more efficient method for delivering inhaled medications to the lungs than the right-angle MDI port (Marik, Hogan & Krikorian, 1999; Mouloudi, Katsanoulas & Anastasaki et al., 1998). The efficacy of MDI drug delivery does not improve with end-inspiratory pause during delivery (Mouloudi et al., 1998) or use of different inspiratory flow pattern during

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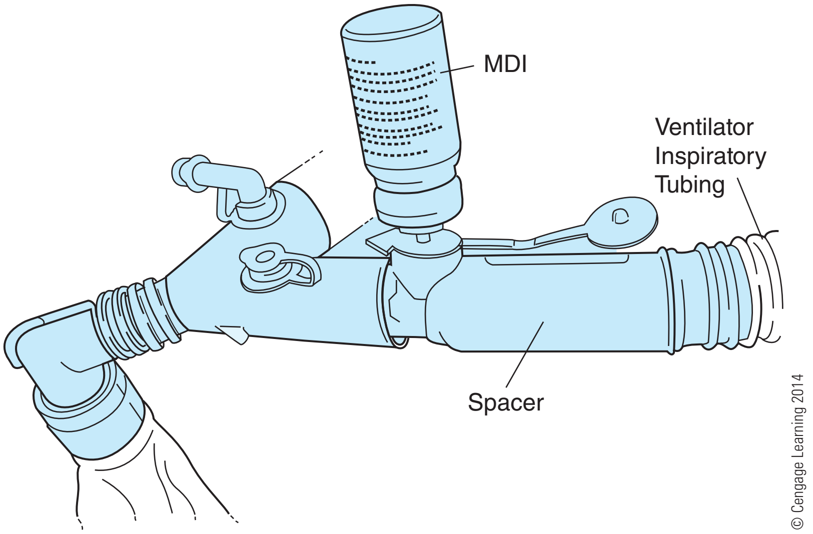

FIGURE 13-2 An MDI with spacer on a ventilator circuit.

pressure-controlled or volume-controlled ventilation (Dhand, 2005). Figure 13-2 shows a typical setup of an MDI on the ventilator circuit.

NEUROMUSCULAR BLOCKING AGENTS

Neuromuscular blocking agents are administered when temporary paralysis of skeletal muscles is desired. Pharmacological paralysis is most commonly induced to (1) ease endotracheal intubation, (2) relieve laryngeal spasm, (3) provide muscle relaxation during surgery, or (4) maintain mechanical ventilation.

Paralyzing agents are used on ventilator patients in difficult situations due to underlying pathology, unnatural modes of ventilation, or psychologic unacceptance. If any of these conditions prevent adequate ventilation, oxygenation, or patient comfort, a paralyzing agent should be considered. The benefits of paralysis during controlled ventilation are given in Table 13-8.

TABLE 13-8 Benefits of Paralysis during Controlled Ventilation

1.	Reduced combativeness and agitation
2.	Relaxation of respiratory muscles
3.	Increased chest wall compliance
4.	Synchronization during unnatural modes of ventilation (e.g., inverse I:E)
5.	Prevention of hypoxemia associated with increased work of breathing
6.	Decreased intracranial pressures caused by excessive movement

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Since serious adverse effects can occur with the use of these agents, they should not be given routinely or before alternative management is considered. If paralysis is indicated, a sedative drug, such as the benzodiazepines along with an opioid analgesic, should be provided for patient comfort. This is necessary because perception and pain thresholds of a patient still exist with use of neuromuscular blocking drugs.

The following testimonies affirm the need for adequate sedation and analgesia during paralysis. One patient who was pharmacologically paralyzed but not sedated described his experience as "a feeling of being buried alive." Another patient thought that she had died (Halloran, 1991). A trauma survivor recalls the sensation of endotracheal tube suctioning being like that of a red-hot burning iron passed into the trachea (Hansen-Flaschen et al., 1993).

Mechanism of Action

During normal neuromuscular transmission, the nerve axon reaches the muscle fibers, and it branches out to form many fine nerve terminals. These nerve terminals are rich in mitochondria, cytoplasmic enzymes, and vesicles. Acetylcholine (ACh), the major chemical in the transmission of nerve impulses, is stored in these vesicles.

When the nerve terminal is stimulated by nerve impulses, acetylcholine is released into the synaptic cleft. From there, some of the acetylcholine is broken down by acetylcholinesterase (ACHe) and other ACh diffuses to the muscle end plate, producing depolarization and muscle contraction. The functional mechanism of neuromuscular transmission is shown in Figure 13-3.

The sequence of events at the neuromuscular junction is as follows. A repeating sequence of depolarization and repolarization is required for continued and

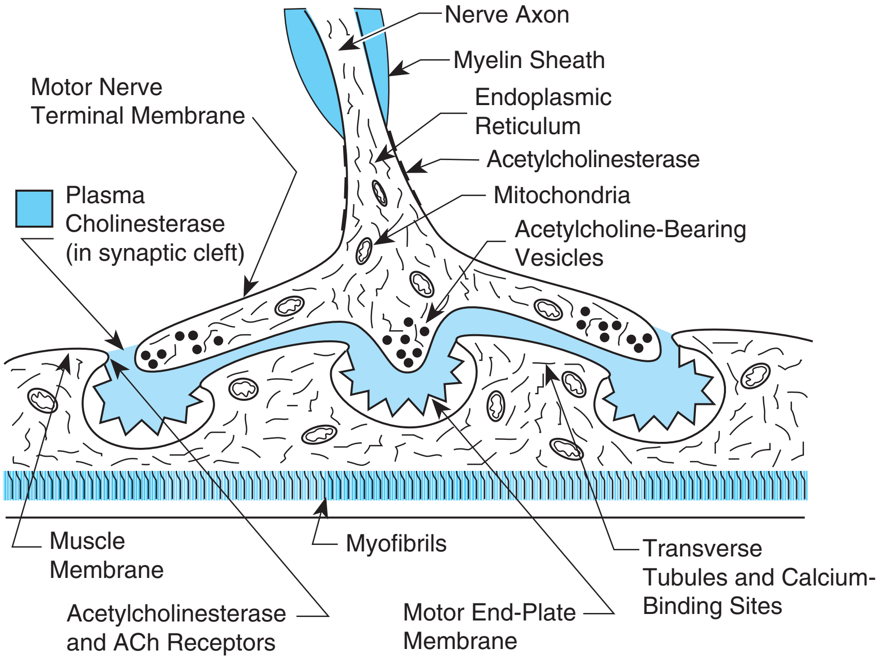
Nerve Axon
Myelin Sheath
Motor Nerve Terminal Membrane
Endoplasmic Reticulum
Acetylcholinesterase
Plasma Cholinesterase (in synaptic cleft)
Mitochondria
Acetylcholine-Bearing Vesicles
Muscle Membrane
Myofibrils
Transverse Tubules and Calcium-Binding Sites
Acetylcholinesterase and ACh Receptors
Motor End-Plate Membrane

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FIGURE 13-3 Functional illustration of neuromuscular transmission.

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coordinated muscular movement. Interruption at any point of the sequence causes muscle relaxation or paralysis, depending on the effective dosage.

Neuromuscular blocking drugs are typically divided into two groups depending on the modes of action at the neuromuscular junction.

depolarizing agents: Drugs that prolong the depolarization phase of muscle contraction, thus rendering the repolarization/depolarization sequence (normal mechanism for muscle movement) impossible and causing muscle blockade. An example is succinylcholine (Anectine, Quelicin).

Depolarizing Agents. The first group of neuromuscular blockers is classified as **depolarizing agents.** This type of agent (e.g., succinylcholine) binds with the receptor site, producing quick onset and sustaining depolarization. Uncoordinated muscle contraction called fasciculation marks the onset. Subsequent neuromuscular transmission is inhibited during the time that adequate concentration of succinylcholine is bound to the receptor site (Ebadi, 1993).

There is no antidote for depolarizing agents. Succinylcholine is, however, rapidly hydrolyzed by plasma pseudocholinesterase (PCHE). A small percentage of the population has an abnormal plasma cholinesterase that does not hydrolyze succinylcholine within minutes, as expected. Plasma PCHE level may also be decreased in-patients with insecticide poisoning, liver disorders (hepatitis, cirrhosis, obstructive jaundice), malnutrition, acute infections, and anemias (Kee, 2005). These individuals may require ventilatory support for hours because of insufficient plasma PCHE.

nondepolarizing agents: Drugs that compete with acetylcholine for the receptor sites at the motor endplates, thus blocking the normal action of acetylcholine and causing muscle blockade. Examples are vecuronium bromide (Norcuron) and pancuronium bromide (Pavulon).

Nondepolarizing Agents. The second group of neuromuscular blockers is classified as **nondepolarizing agents.** These agents (e.g., Norcuron and Pavulon) compete with acetylcholine for the receptor sites at the motor endplates, thus blocking the normal action of acetylcholine. Since the nondepolarizing agents compete for the receptor sites, they are also called competitive agents. They are antagonized by anticholinesterase agents such as pyridostigmine and neostigmine. Anticholinesterase agents allow ACh levels to rise and reverse the effects of nondepolarizing agents.

Characteristics of Neuromuscular Blocking Agents

Pharmacologically induced blockade progresses in the following sequence: rapidly contracting muscles (eyes and digits) followed by larger and slower contracting muscles (extremities, trunk, and diaphragm) (Halloran, 1991). Depolarizing agents (e.g., succinylcholine) have a quick onset but are short-lasting, making them the drugs of choice for emergency intubation. Nondepolarizing agents have longer onsets ranging from 3 to 10 min, but are longer lasting. These drugs are more appropriate for controlled ventilation in the intensive care unit. Table 13-9 shows the characteristics of selected depolarizing and nondepolarizing neuromuscular blocking agents.

Factors Affecting Neuromuscular Blockade

Several factors can alter neuromuscular transmission and blockade. They include organ failure, drug interaction, electrolyte imbalance, and acid-base status.

Organ Failure. Patients with altered renal or hepatic function have an increased risk of prolonged blockade. These patients can remain profoundly weak long after the drug is discontinued. Both pancuronium bromide and vecuronium bromide have

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TABLE 13-9 Depolarizing and Nondepolarizing Agents

Agents	Initial Dose (mg/kg)	Onset (min)	Duration	Drug Clearance
Depolarizing				
Succinylcholine (Anectine, Quelicin)	0.3 to 1.1	1	Short	Metabolized by plasma cholinesterase
Nondepolarizing				
Pancuronium bromide (Pavulon)	0.07	5 to 7	Long	Metabolized by liver, excreted in urine
Atracurium (Tracrium)	0.4 to 0.5	4 to 5	Intermediate	Self-destroying
Vecuronium bromide (Norcuron)	0.05	4 to 6	Intermediate	Metabolized by liver, excreted in urine
Rocuronium (Zemuron)	0.3	3 to 4	Short	Metabolized by liver

✓ Organ failure may decrease drug clearance, increase drug accumulation, and prolong neuromuscular blockade.

been implicated in drug accumulation in patients with renal insufficiency. Vecuronium is also poorly eliminated in patients with hepatic failure. Atracurium is preferred in the presence of organ failure because it is self-destroying and does not rely on organ metabolism and excretion (Halloran, 1991).

✓ Beta blockers, procainamide, quinidine, calcium channel blockers, and nitroglycerin may potentiate the effects of nondepolarizing agents.

Drug Interaction. Some cardiovascular and antiarrhythmic agents may interact and potentiate the effects of nondepolarizing agents. They include beta blockers, procainamide, quinidine, calcium channel blockers, and nitroglycerin. High concentrations of antibiotics may also potentiate the effects of competitive agents by decreasing the release of ACh. These antibiotics include the aminoglycosides and polymyxins as well as tetracycline, erythromycin, and vancomycin (Halloran, 1991).

Steroidal-based vecuronium bromide and pancuronium bromide may be particularly dangerous if administered along with systemic corticosteroids (Watling et al., 1994). A significant number of asthmatics receiving these neuromuscular blocking agents in combination with steroid therapy have experienced prolonged blockade for several days following discontinuation of the paralyzing agent (Kupfer et al., 1987). This prolonged blockade is possibly related to the comparable chemical structure of steroid and steroidal-based agents; however, the mechanism is unknown. Figure 13-4 shows the basic steroid structure and pipecuronium, a steroidal-based paralyzing agent. Until studies become conclusive, use of corticosteroids should be avoided during prolonged muscle blockade with any neuromuscular blocking agent (Hansen-Flaschen et al., 1993).

✓ ↑ Ca^{++} may lead to ↑ release of ACh and ↑ muscular contraction.

Electrolyte Imbalance. The normal physiology of muscle contraction depends on the regulation of electrolytes. Abnormal levels of calcium and magnesium affect the quality of contraction, while imbalances in potassium and sodium levels alter the excitability at

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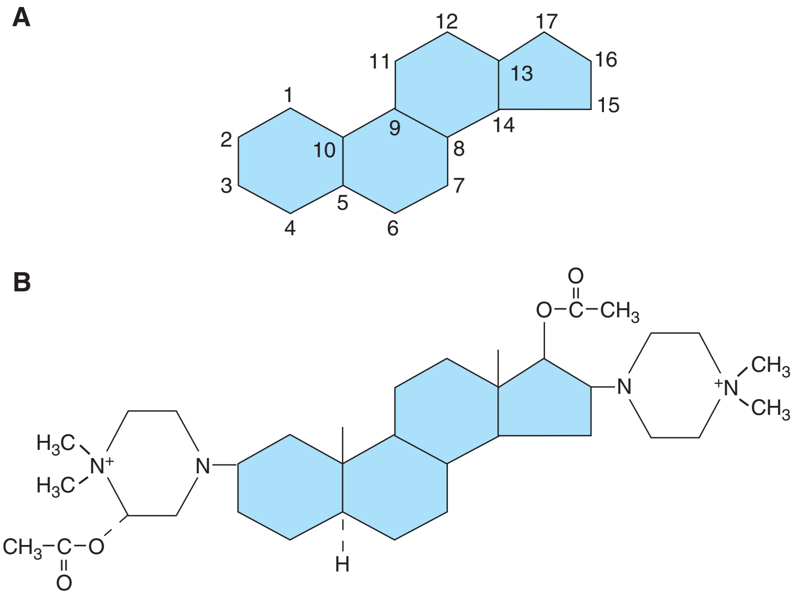

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FIGURE 13-4 (A) Basic steroid structure; (B) A steroidal-based neuromuscular blocking agent, pipecuronium.

↑ Mg^{++} may lead to ↓ release of ACh and ↓ muscular contraction.

↓ Ca^{++} or ↑ Mg^{++} enhances neuromuscular blockade with *nondepolarizing* agents.

↑ Mg^{++} enhances neuromuscular blockade with *depolarizing* agents.

Diuretic-induced hypokalemia causes ↑ blockade with nondepolarizing agents and ↓ blockade with depolarizing agents.

the motor endplate. For example, the release of ACh depends on extracellular calcium and magnesium concentrations. Calcium functions to release ACh from the vesicles and expose myosin-binding sites on actin. Myosin-binding site exposure is a structural change that is necessary for the sliding of thin myofilaments past thick myofilaments. The sliding of these filaments results in muscular contraction (Tortora et al., 2002). Magnesium works in opposition to calcium. It decreases the release of ACh as well as the membrane's sensitivity, thus inhibiting muscle contraction (Ebadi, 1993).

Consequently, low calcium and high magnesium levels can enhance the effects of nondepolarizing agents. Increased magnesium levels can magnify the effects of depolarizing agents.

Sodium and potassium have a major function in the process of depolarization. When a muscle cell is at rest, or not depolarized, there is a considerable difference between the concentration of sodium and potassium outside and inside the plasma membrane. At rest, there is an increased concentration of sodium extracellularly and an increased concentration of potassium intracellularly. The difference in charge on either side of the membrane of the resting cell is known as the resting potential and is said to be polarized or charged (Tortora et al., 2002).

An acute decrease in extracellular potassium will result in hyperpolarization and an increased resistance to depolarization. Hence, hypokalemia augments nondepolarizing agents and antagonizes depolarizing agents (Ebadi, 1993). Diuretic-induced hypokalemia should be corrected to prevent altered effects (potentiation of blockade with nondepolarizing agents; reduction of blockade with depolarizing agents) (Halloran, 1991).

Acid-Base Status. Acidemia intensifies neuromuscular blockade, requiring a lower dosage of paralyzing agent or a higher dosage of reversal agent such as neostigmine (Kupfer et al., 1987). On the other hand, alkalemia necessitates a higher dosage of

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✓ Acidemia intensifies the effects of neuromuscular blockade.

✓ Alkalemia diminishes the effects of neuromuscular blockade.

paralyzing agent to maintain neuromuscular blockade. These alterations are likely the result of potassium shift (extracellularly) associated with acidemia. H^+ moves into cells to be buffered, and K^+ moves out to maintain ionic neutrality. One might find these effects to be significant when intracranial pressure is being reduced via hyperventilation (respiratory alkalosis).

Although the preceding factors can alter the action of neuromuscular blocking agents, the net effects following the initial dosage can be titrated based on the results of patient monitoring. Since there is no standard level of paralysis to be achieved, the desired depth of blockade will depend on the clinical objectives of the physician.

Adverse Effects

Apnea is the most immediate and life-threatening adverse effect associated with both depolarizing and nondepolarizing agents. For this reason, practitioners experienced in airway management must be present when these drugs are administered. For a patient who is already intubated and committed to a mechanical ventilator, the following alarms should be active.

- Apnea alarm
- Low pressure/disconnect
- Low exhaled tidal/minute volume
- High/low heart rate
- Low SpO_2

Deaths due to apnea have been reported in cases where alarms were inappropriately set or inactivated. It is possible that the incidence of fatal outcome is underreported (Halloran, 1991).

Other undesirable effects of neuromuscular blocking agents include loss of cough mechanism, blunted neurologic assessment, emotional trauma due to inadequate sedation, disuse muscle atrophy, pressure sores, and increased iatrogenic morbidity and mortality associated with extended ICU exposure.

✓ Succinylcholine and atracurium may provoke bronchospasm and hypotension due to histamine release.

Histamine release is a property shared by most nondepolarizing agents. The presence of histamine may be manifested clinically as vasodilation, flushing, and bronchospasm. The degree of histamine release differs among the drugs. Succinylcholine, tubocurarine, metocurine, and atracurium have been known to provoke bronchospasm and hypotension related to moderate histamine release, whereas pancuronium elicits only minimal release. Histamine release is not likely with the use of vecuronium (Ebadi, 1993).

Cardiovascular effects range from minimal to moderate, including bradycardia, tachycardia, arrhythmias, and circulatory collapse. Sudden changes in drug levels associated with intermittent administration (versus continuous infusion) seem to be responsible for these adverse effects (Watling et al., 1994). Table 13-10 summarizes the bronchopulmonary and cardiovascular adverse effects of neuromuscular blocking agents.

A rare but very serious hazard of succinylcholine and inhaled anesthetics is a condition known as malignant hyperthermia (MH). In individuals with the genetic predisposition, MH causes skeletal muscle metabolism to suddenly surge out of control,

TABLE 13-10 Adverse Effects of Neuromuscular Blocking Agents

Agents	Histamine Release	Cardiovascular Impairment	Clinical Considerations
Depolarizing			
Succinylcholine (Anectine, Quelicin)	Moderate	Moderate	Caution with plasma cholinesterase disorder
Nondepolarizing			
Pancuronium bromide (Pavulon)	Minimal	Moderate	Steroidal-based Use corticosteroids with caution
Atracurium (Tracrium)	Moderate	Minimal	Not affected by organ dysfunction
Vecuronium bromide (Norcuron)	Not likely	Minimal	Steroidal-based Use corticosteroids with caution
Rocuronium (Zemuron)	Minimal	Minimal	Use corticosteroids with caution

depleting oxygen, generating excessive carbon dioxide, spiking body temperature, and causing circulatory collapse and death if not treated immediately. The fastest way to detect MH is by monitoring with capnography (to detect rapid increase in exhaled CO_2) when administering succinylcholine and volatile anesthetics. The preferred treatment for MH is dantrolene sodium (Halsall et al., 2003).

Evaluation of Neuromuscular Blockade

To prevent unintentional overdosing, clinicians must establish an objective method of monitoring the depth of paralysis. This is especially meaningful in the management of patients with potential for drug accumulation secondary to renal or hepatic dysfunction.

A peripheral nerve stimulator is a valuable tool used to monitor the degree of neuromuscular blockade in patients who are pharmacologically paralyzed. It can measure the degree of blockade by measuring the number of muscle twitches in response to four sequential stimuli delivered over a two-second period. This is called a Train-of-Four (ToF) stimulus. Two electrodes are placed along a nerve path where electrical stimuli are delivered at a frequency of 2 Hz (four times in 0.5-sec intervals). As the degree of blockade increases, the number of elicited responses (muscle twitches) decreases. The ulnar, facial, and posterior tibial nerves are commonly used because they are superficial and easy to locate. Figure 13-5 shows the electrode placement along the ulnar nerve.

Most recommendations for ToF monitoring suggest titration of neuromuscular blocker to one or two twitches ($>$80% to 90% muscular blockade) in 2 sec, which is the current practice in using ToF monitoring. However, ToF of three twitches generally corresponds closely to 80% muscular blockade. This lighter level of muscular blockade may be adequate to assure patient-ventilator synchrony and lower airway pressures and to optimize oxygen delivery in most patients (Strange et al., 1997).

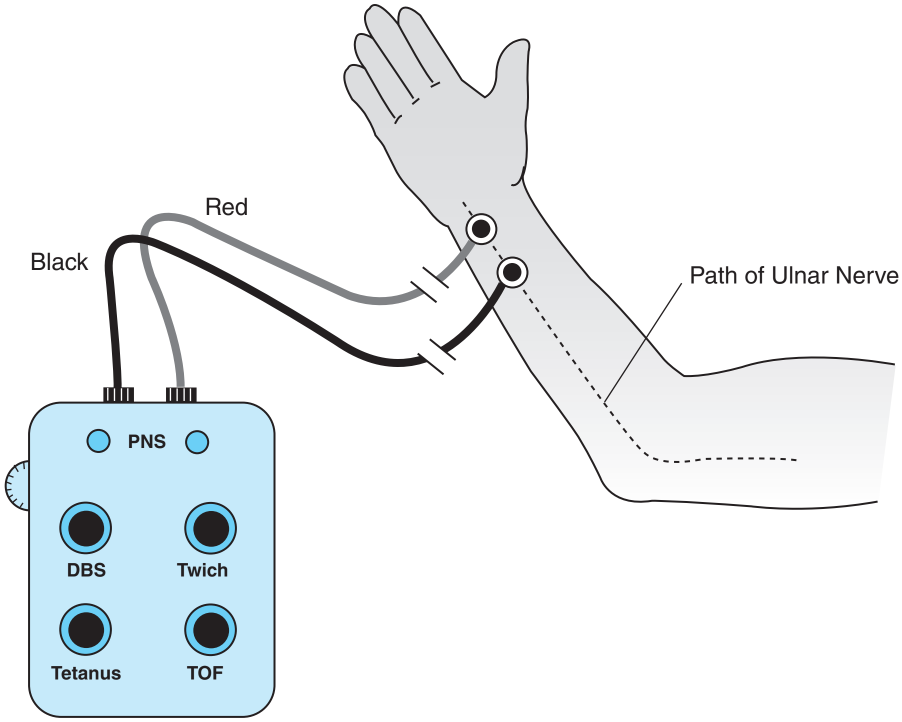

FIGURE 13-5 Placement of the Train-of-Four electrodes along the ulnar nerve.

A clinically useful but less objective method of measuring the depth of blockade is by assessing the patient's spontaneous muscle effort. Recovery of muscle blockade occurs in a reverse sequence. The ability to open one's eyes widely, sustain head lift, and protrude the tongue for more than 5 sec confirms adequate reversal. Ventilator support, however, should not be discontinued until the diaphragm is able to provide adequate ventilation. Arterial blood gases and spontaneous maneuvers (e.g., maximal inspiratory pressure [MIP] and vital capacity [VC]) can provide evidence of partial or full diaphragm recovery (Halloran, 1991). Table 13-11 outlines the method for assessment of neuromuscular blockade reversal.

✓ The ability to open eyes widely, sustain head lift, and protrude the tongue for more than 5 sec confirms adequate reversal of neuromuscular blockade.

TABLE 13-11 Assessment of Neuromuscular Blockade Reversal

1. Head lift 5 sec
2. Tongue protrusion 5 sec
3. Hand grip
4. Arterial blood gases with $PaO_2 > 80$ mm Hg and $PaCO_2 < 45$ mm Hg*
5. Maximal inspiratory pressure (MIP) at least −25 cm H_2O
6. Vital capacity greater than 900 mL

**Slightly higher $PaCO_2$ is acceptable for patients with chronic CO_2 retention.*

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CENTRAL NERVOUS SYSTEM AGENTS

Medications that act on the central nervous system are often categorized by their primary effect, but this can create problems when the substance has multiple effects (which is the case for many). It is less confusing to categorize a drug by its composition rather than by one of its therapeutic effects. For instance, a benzodiazepine can produce sedation to the point of sleep (hypnotic sedative), anxiety relief (anxiolytic), amnesia, muscle relaxation, and relief from seizures. Ketamine is an unusual drug in that it has both analgesic and sedation properties. Common terms for central nervous system effects include anesthesia (diminished bodily sensation), analgesia (relief of pain), sedation (diminished awareness or consciousness), and neurolepsis (a particular alteration of consciousness that produces reduced anxiety, calming, and indifference to one's surroundings). The central nervous system (CNS) medications in the following sections are organized and shown in Figure 13-6. Although inhaled anesthetics are typically reserved for the operating suite and not discussed in this chapter, it is worth noting that these agents are sometimes used in the emergency department to treat extreme exacerbations of asthma.

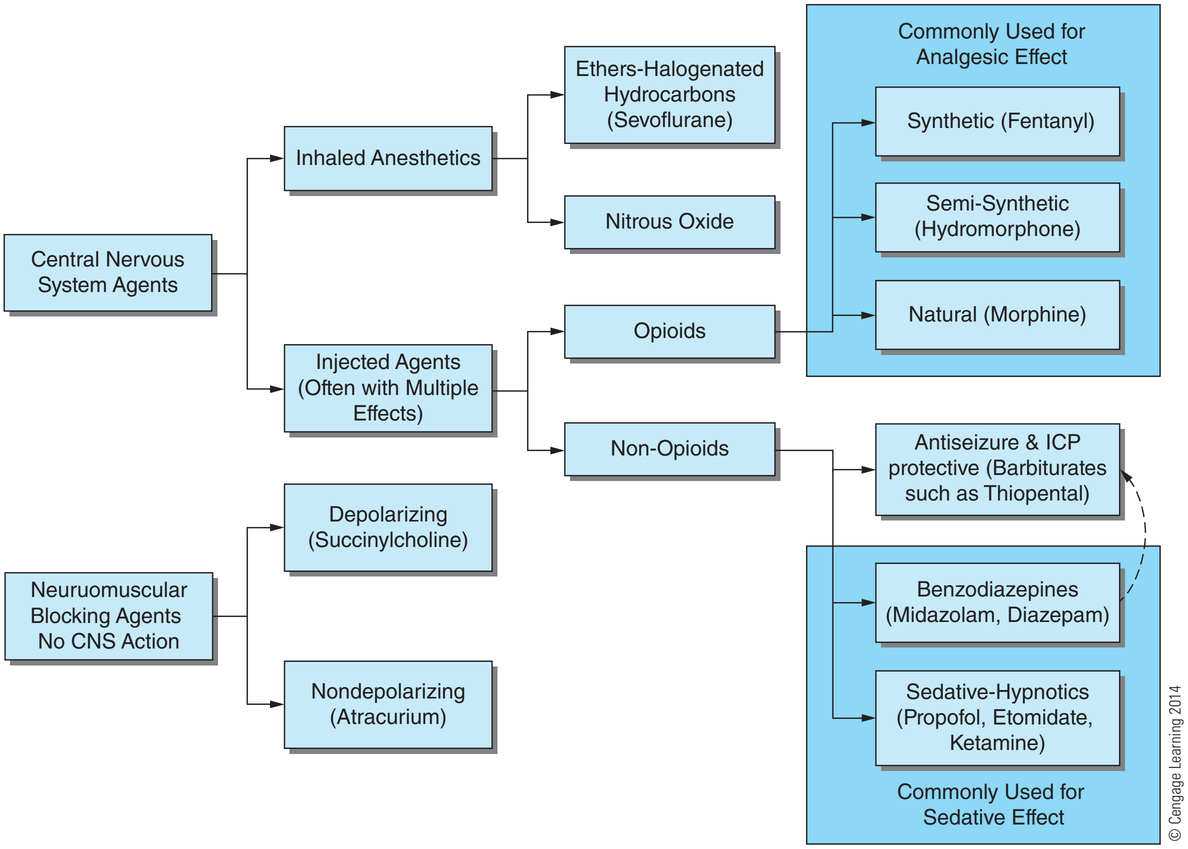
Central Nervous System Agents
Inhaled Anesthetics
Ethers-Halogenated Hydrocarbons (Sevoflurane)
Nitrous Oxide
Injected Agents (Often with Multiple Effects)
Opioids
Non-Opioids
Commonly Used for Analgesic Effect
Synthetic (Fentanyl)
Semi-Synthetic (Hydromorphone)
Natural (Morphine)
Antiseizure & ICP protective (Barbiturates such as Thiopental)
Benzodiazepines (Midazolam, Diazepam)
Sedative-Hypnotics (Propofol, Etomidate, Ketamine)
Commonly Used for Sedative Effect
Neuruomuscular Blocking Agents No CNS Action
Depolarizing (Succinylcholine)
Nondepolarizing (Atracurium)
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FIGURE 13-6 Examples of drugs that exert their effects on the central nervous system.

Sedatives and Antianxiety Agents (Benzodiazepines)

benzodiazepines: A group of drugs with strong hypnotic and sedative actions; used mainly to reduce anxiety and to induce sleep.

Benzodiazepines are given to patients to reduce anxiety, provide amnesia, and improve tolerance of mechanical ventilation. In addition, they facilitate invasive procedures (e.g., bronchoscopy, intravenous access, suctioning), decrease oxygen consumption, and protect the patient from self-injury.

Mechanism of Action. Binding receptors for benzodiazepines have been identified in the limbic, thalamic, and hypothalamic levels of the central nervous system (Mohler et al., 1977). These receptor sites appear to be involved in a large molecular complex that includes **gamma-aminobutyric acid (GABA)** receptors and GABA-regulated chloride ion channels (Figure 13-7).

gamma-aminobutyric acid (GABA): A major central nervous system inhibitory transmitter that regulates the chloride ion channel and hyperpolarizes the neurons. Once the neurons are hyperpolarized and become resistant to repeated depolarization, sedation results.

GABA Mechanism. GABA is a major central nervous system inhibitory transmitter that opens chloride ion channels. Once hyperpolarized by the GABA action, the neurons become more resistant to repeated depolarization and sedation results (Mohler et al., 1988). Benzodiazepines facilitate the action of GABA, thus producing clinical sedation, **anxiolysis,** anticonvulsant effects, amnesia, slowing of reaction time, visual accommodation difficulties, and ataxia.

anxiolysis: Diminishing anxiety.

✓ Benzodiazepines are normally well absorbed in the gastrointestinal (GI) tract. In unstable patients, GI tract absorption may be unreliable, and parenteral administration is preferred.

Absorption. Benzodiazepines are normally well absorbed in the gastrointestinal (GI) tract. In unstable patients, GI tract absorption may be unreliable, and parenteral administration is preferred. Lorazepam and midazolam are rapidly and completely absorbed intramuscularly, whereas the absorption of chlordiazepoxide and diazepam is slow and erratic (McEvoy, 1995). Because of the slower onset of action and pain associated with intramuscular administration, the intravenous route is preferred in the critically ill. The lipid solubility of the drugs allows rapid distribution across the blood-brain barrier with midazolam being the most lipophilic, followed by diazepam and chlordiazepoxide, and then lorazepam (McEvoy, 1995).

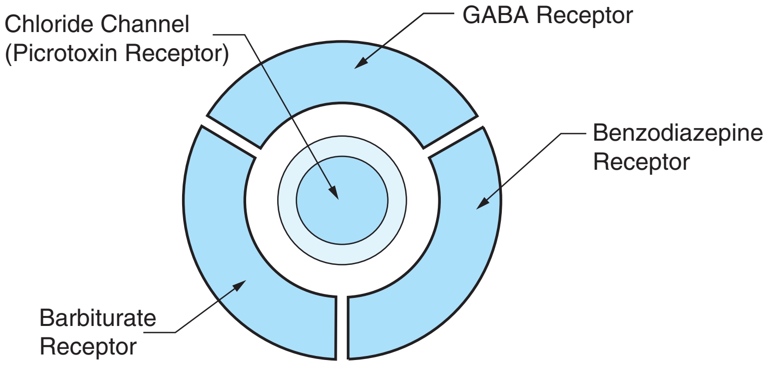

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FIGURE 13-7 Gamma-aminobutyric acid (GABA) receptor complex. GABA is the most common inhibitory neurotransmitter in the central nervous system (CNS). Activation of the postsynaptic GABA receptor increases chloride conductance through the ion channel thus hyperpolarizing it and inhibiting the postsynaptic neuron. Once the postsynaptic neurons are inhibited, depression of the CNS and neuromuscular blockade results.

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Excretion. Benzodiazepines are metabolized in the liver to active and inactive metabolites that are excreted mainly in the urine. Although the pharmacokinetics of drugs are often used to explain correlations between plasma concentrations and effect, benzodiazepines do not exhibit this relationship. Furthermore, carefully controlled clinical trials have not shown the superiority of one agent over another for various ICU indications (Dasta et al., 1994). Therefore, the choice of benzodiazepine is often based on cost.

✓ Dose-dependent adverse CNS effects include confusion, weakness, dizziness, drowsiness, ataxia, syncope, vertigo, and amnesia.

✓ Withdrawal syndrome (anxiety, tachycardia, diaphoresis, hypertension, and, occasionally, seizures) may occur if benzodiazepines are abruptly discontinued.

✓ Benzodiazepines may decrease the mean arterial pressure, stroke volume, cardiac output, and systemic vascular resistance.

Adverse Effects. Dose-dependent adverse central nervous system effects are very common with benzodiazepines. They are an extension of the pharmacologic actions of the drugs and include confusion, weakness, dizziness, drowsiness, ataxia, syncope, vertigo, and amnesia (McEvoy, 1995). Central nervous system stimulation occurs occasionally in patients with underlying psychiatric disorders. These patients present with restlessness, mania, euphoria, acute rage reactions, and sleep disorders (McCartney et al., 1993). A withdrawal syndrome may occur if these drugs are abruptly discontinued in patients on prolonged therapy or high doses. This may be manifested by severe anxiety, tachycardia, diaphoresis, hypertension, and, occasionally, seizures (McCartney et al., 1994).

Parenteral administration of benzodiazepines may result in a dose-dependent respiratory depression (Forster et al., 1980) which is additive to that produced by opioids. Elderly and COPD patients are at the greatest risk (Levine, 1994). Although depression of the respiratory drive is not usually as great with benzodiazepines as with other sedative agents, apnea may occur. Careful dosing of these agents is also necessary to avoid prolonged mechanical ventilation.

Benzodiazepines may also have direct effects on cardiovascular hemodynamics. They have been shown to decrease mean arterial pressure, stroke volume, cardiac output, and systemic vascular resistance (Reves et al., 1990). Additive decreases in blood pressure may occur when opioids are administered concurrently. Table 13-12 outlines the cardiovascular effects of benzodiazepines.

Assessment of Sedation. The indication for benzodiazepines and the estimated duration of therapy may be helpful in deciding whether to administer these drugs intermittently or continuously. Sedation and analgesia protocols are recommended along with daily sedation holidays to avoid prolonging a patient's requirement for

TABLE 13-12 Cardiovascular Effects of Benzodiazepines

1.	↓ Mean arterial pressure
2.	↓ Stroke volume
3.	↓ Cardiac output
4.	↓ Systemic vascular resistance
5.	↓ Blood pressure

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TABLE 13-13 Ramsay Scale for Assessment of Sedation

Level/Score	Clinical Description
I	Anxious and agitated
II	Cooperative, oriented, tranquil
III	Responds only to verbal commands
IV	Asleep with brisk response to light stimulation
V	Asleep with sluggish response to stimulation
VI	Asleep without response to stimulation

mechanical ventilation. If a short period of sedation is needed, intermittent intravenous bolus dosing may prevent oversedation. If prolonged periods of sedation are required, a bolus followed by a continuous infusion may provide optimal patient comfort. If the indication is to facilitate an uncomfortable procedure, an intravenous bolus dose should be given.

Since individual patient response to benzodiazepines is highly variable, monitoring is essential to ensure correct dosing and reduce costs. Assessment of a patient's sedation state may be done with the **Ramsay Scale** for Assessment of Sedation as outlined in Table 13-13 (Ramsay et al., 1974). Using the Ramsay Scale and the appropriate types and dosages of benzodiazepines (Table 13-14), a desired level of sedation may be determined.

It is important to note that the Ramsay Scale is not suitable for paralyzed patients. Since they cannot perform those required commands for the Ramsay Scale, autonomic signs such as tachycardia, diaphoresis, hypertension, and lacrimation may suggest inadequate sedation or pain control (Shelly et al., 1986).

Ramsay Scale: The scoring system with a scale ranging from level I to level VI; used to assess the degree of sedation.

✓ Ramsay Scale is not suitable for paralyzed patients since they cannot perform those required commands.

✓ Autonomic signs (tachycardia, diaphoresis, hypertension, and lacrimation) may suggest inadequate sedation or pain control.

Opioid Analgesics

Patients on mechanical ventilation may have a variety of medical or surgical problems that necessitate attention to pain control. In these patients, however, pain is difficult

TABLE 13-14 Common Benzodiazepines Used in Mechanical Ventilation

Drug	Initial Dose	Onset	Duration
Diazepam (Valium)	2.5 to 10 mg IV	Fast	Short initially; multiple doses result in prolonged effect
Lorazepam (Ativan)	0.5 to 2 mg IV	Intermediate	Intermediate
Midazolam (Versed)	1 to 5 mg IV	Fast	Short, but may be prolonged if not carefully dosed

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TABLE 13-15 Adverse Patient Outcomes Associated with Pain

Reaction Induced by Pain	Adverse Outcomes
Tissue initiated stress hormone response	Breakdown of body tissue Increased blood clotting Increased metabolic rate Increased water retention Decreased immune function
Activation of autonomic functions	Increased blood pressure Increased heart rate
Muscle splinting	Decreased tidal volume Decreased respiratory rate Decreased minute ventilation
Immobility	Formation of deep vein thrombosis and pulmonary embolism
Diminished gastrointestinal function	Delay of bowel and gastric function

to assess because of their inability to speak. Hemodynamic and respiratory instability, and immediate life-threatening concerns also overshadow the presence of pain. Subsequently, pain control for these patients is often inadequate (Wheeler, 1993).

Pain can cause anxiety, discomfort, and delirium in patients on mechanical ventilation. Interference with sleep also compounds the level of anxiety, thus further elevating the perception of pain. Pain is also associated with many adverse outcomes in the intensive care unit (Table 13-15). **Opioid analgesics** are commonly used to control pain to allow earlier patient mobilization and to decrease hospital stay.

opioid analgesics: Drugs that are used to control pain via the central nervous system. Examples are morphine, codeine, and meperidine (Demerol, a synthetic compound).

Mechanism of Action. Opioid analgesics produce analgesia by binding to opioid receptors in and outside the central nervous system. Several subtypes of opiate receptors have been identified. The mu, kappa, and sigma receptors are known to have important nociceptive (neural receptors for painful stimulus) properties in humans. Binding and activating these receptors also help explain the adverse pharmacologic profile of these drugs (Table 13-16) (Teeple, 1990).

Opiates may be further classified depending on whether they are agonists, agonist-antagonists, or pure antagonists at these receptors (Table 13-17). Full agonists produce a maximal response within cells to which they bind; agonist-antagonists activate one type of opiate receptor while blocking another; and antagonists only block opiate receptors (Teeple, 1990).

✓ Full agonists produce a maximal response within cells to which they bind.

✓ Agonist-antagonists activate one type of opiate receptor while blocking another.

Table 13-17 contains only representative samples from each class. It is not a complete listing of all opioids.

Most agonist-antagonist drugs are agonists at the sigma receptor. As the dose of these drugs is increased for deeper analgesia, the more likely they may cause significant psychological effects such as delirium. This class of drugs is not routinely used in ventilator patients and will not be discussed further.

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TABLE 13-16 Narcotic Receptor Classifications

Physical Parameter	mu	kappa	sigma
Pain sensation	−	−	0
Body temperature	−	0	+
Pulse rate	−	0	+
Pupils	−	−	+
Respiratory rate	−	−	+
CNS effect	Sedation	Hypnosis	Delirium

0 *no effect*
+ *increased effect*
− *reduced effect*

TABLE 13-17 Opioid Drug Classifications

Drugs [Selected Examples]	mu	kappa	sigma
1. Agonist [morphine, meperidine (Demerol), fentanyl (Sublimaze)]	+	+	0
2. Agonist-antagonist [pentazocine, butorphanol]	−	+	+
3. Antagonist [naloxone (Narcan)]	−	−	−

0 *no effect*
+ *agonist*
− *antagonist*

✓ Antagonists block opiate receptors.

Antagonist drugs such as naloxone (Narcan) are primarily used to reverse overdose of narcotics. Special care must be exercised with the use of naloxone in a patient being treated for severe pain. It may reverse an untoward adverse effect such as respiratory depression while causing the return of severe pain (Levine, 1994).

Adverse Effects. Opioid analgesics produce many adverse effects. Table 13-18 outlines the adverse effects that may occur to various systems or body locations of the patient.

✓ Sedation, respiratory depression, and shallow breathing are the primary adverse effects of opioid analgesics on the CNS.

Central Nervous System. Opiates cause sedation, respiratory depression, and myoclonus (twitching or spasm of muscles) and their effects are dose-dependent (Levine, 1994). Clinically significant respiratory depression is more likely to occur with a large initial dose or inpatients with underlying pulmonary disease. Respiratory depression (decreased tidal volume and frequency) is mediated by opioid agonist activity at the mu receptors of the brain stem (Teeple, 1990). Since opiates suppress deep breathing, they may induce atelectasis, which can be prevented by activating the automatic sigh mode on the ventilator.

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TABLE 13-18 Adverse Effects of Narcotic Analgesics

Location	Adverse Effects
Central nervous system	Sedation Respiratory depression Shallow breathing (and atelectasis)
Muscle groups	Myoclonus (twitching or spasm of muscles) Convulsions Chest wall rigidity
Cardiovascular	Direct vasodilation Vagally mediated bradycardia Hypotension
Gastrointestinal	Delayed gastric emptying Constipation Nausea
Others	Miosis (contraction of pupils) Altered levels of stress hormones Uncommon allergic reactions

✓ Myoclonus (twitching or spasm of muscles), convulsions, and chest wall rigidity are the primary adverse effects of opioid analgesics on the muscle group.

Muscle Groups. Myoclonus (twitching or spasm of muscles) and other neuroexcitatory phenomena have been reported with opioid analgesics. Myoclonus may be prevented by switching to another opioid, lowering the dosage, or using a benzodiazepine (Valium), an antianxiety and hypnotic agent. Convulsions have been reported with high doses of any opioid in an intolerant patient, but are most common with normeperidine—a liver metabolite of meperidine (Demerol) (Brucera et al., 1992). Naloxone (Narcan) may be used to reverse convulsions caused by opiates with the exception of those caused by the use of meperidine.

Chest wall rigidity is a complication that may develop after administration of any opiate, but it is most commonly reported with fentanyl. This adverse effect is most often seen at the time of anesthesia induction or after surgery. It may be so severe that the patient may require intubation, mechanical ventilation, and chemical paralysis. Patients at risk for this untoward effect appear to be the elderly, patients with renal failure, and those receiving large doses of opioids (Wheeler, 1993).

✓ Direct vasodilation, vagally mediated bradycardia, and hypotension are the primary adverse effects of narcotic analgesics on the cardiovascular system.

Cardiovascular Effects. Opioids can affect a patient's hemodynamic status. Hypotension may develop as a result of direct vasodilation, histamine release, and vagally mediated bradycardia (Levine, 1994). These complications may be prevented by using the lowest effective dose, providing an adequate intravascular volume, or decreasing the rate of administration. (Note: Meperidine's [Demerol's] structure resembles that of atropine; thus it may cause tachycardia rather than bradycardia.)

Although opioids may produce a dose-dependent clinical spectrum ranging from pain relief to sedation, deep coma, and anesthesia, low-dose opiates are often combined with low-dose sedatives (benzodiazepines) to minimize the adverse effects of these two agents.

Gastrointestinal Effects. The gastrointestinal effects of opioids include delayed gastric emptying, constipation, and nausea (Levine, 1994). Since tolerance to constipation does not occur, or occurs very slowly during opiate administration, **cathartic agents** should be given on a regular basis to activate bowel movements.

cathartic agents: Active purgatives used to produce bowel movements.

✓ Delayed gastric emptying, constipation, and nausea are the primary adverse effects of narcotic analgesics on the GI system.

Nausea and vomiting during opiate administration may be due to three different mechanisms. First, opiates may reduce gastric motility that results in nausea after eating. Second, opioids seem to cause nausea that is due to sensitization of the vestibular apparatus. This type of nausea is brought on by changes in position or head movement. Third, these drugs may directly stimulate the medullary chemoreceptor trigger zone. Nausea from this mechanism is usually present continuously (Jacox et al., 1994).

Other adverse effects related to opioid use include miosis (contraction of pupils), altered levels of stress hormones, and uncommon allergic reactions.

Clinical Considerations. Opioid tolerance, physical dependence, and psychological dependence are important concepts to understand when considering analgesic therapy. Misuse of these terms has led to ineffective practices in prescribing, administering, and treatment of patients in pain.

Tolerance. Tolerance is defined as the need to increase dosage requirements to maintain effective pain relief. In addition, tolerance may occur to some adverse opioid effects such as respiratory depression, miosis, sedation, and nausea (Foley, 1993).

Physical Dependence. Physical dependence is defined as the precipitation of a withdrawal syndrome upon abrupt termination of the drug or after administration of a narcotic antagonist (naloxone). Clinically, the withdrawal symptoms include irritability, joint pain, chills and hot flashes, anxiety, nausea, vomiting, lacrimation, rhinorrhea, diaphoresis, abdominal cramps, and diarrhea. Withdrawal symptoms may be avoided in physically dependent patients by gradual dosage reduction of the opiate (Hammack et al., 1994).

Psychological Dependence. Psychological dependence is an addictive behavior characterized by drug seeking, preoccupation with obtaining and using the drug, and drug use for other than analgesic purposes (euphoria).

✓ Since ventilator patients cannot communicate effectively, these clinical signs may be used to reflect inadequate pain control.

Assessment of Adequate Pain Control. Pain assessment is important in ensuring adequate pain relief and enhancing patient recovery. Cooperative, awake, and alert patients may be assessed with pain intensity and pain distress scales. Unfortunately, ventilator patients are frequently unable to participate in their pain management plan. In this case, clinical signs such as tachycardia, blood pressure changes, dilated pupils, diaphoresis, grimacing, restlessness, and guarding may be signs that indicate inadequate analgesia (Table 13-19).

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TABLE 13-19 Clinical Signs of Inadequate Pain Control

Tachycardia in the absence of hypoxemia
Blood pressure changes
Dilated pupils
Diaphoresis
Grimacing
Restlessness
Guarding

Agents for Seizures and Elevated Intracranial Pressure (Barbiturates)

barbiturates: A group of drugs that depresses the central nervous system. Adverse effects are many, including alteration of the respiration, heart rate, blood pressure, and temperature. They are used in seizure disorders, control of elevated intracranial pressure.

Barbiturates, once widely used in critically ill patients for sedation and anxiolysis, have limited applications in patients on mechanical ventilation because of respiratory and cardiovascular depression. For sedative action, they have been replaced by safer drugs such as benzodiazepines. Barbiturates may be preferred in certain situations, such as seizure disorders, control of elevated intracranial pressure (normal ICP 8 to 12 mm Hg, $<$20 mm Hg in clinical practice).

Table 13-20 lists the selected barbiturates that are used in different situations because of the wide range of duration of action, ranging from ultrashort-acting (0.2 hour) to long-lasting (4 to 12 hours).

TABLE 13-20 Selected Barbiturates

Names	Duration of Action	Duration of Hypnotic* Effect
Thiopental Methohexital	Ultrashort-acting	0.2 hour
Pentobarbital	Short-acting	1 to 4 hours
Amobarbital	Intermediate-acting	2 to 8 hours
Phenobarbital	Long-acting	4 to 12 hours

**The anesthetic dose is about three times the hypnotic dose; the lethal dose is about six times the hypnotic dose (Cottrell et al., 1995; Ziment, 1978).*

Mechanism of Action. Barbiturates are capable of producing all levels of CNS depression, ranging from mild sedation to hypnosis to deep coma to death. As previously discussed in the benzodiazepine section, GABA is the most common CNS inhibitory neurotransmitter. The GABA receptor exists as a complex involving the benzodiazepine receptor, the barbiturate receptor, and the GABA receptor and its associated chloride ion channel (Figure 13-7) (Olson, 1988). Drug binding to this receptor complex facilitates GABA-mediated hyperpolarization of the neuron via an increase in chloride ion entry into the cell. This hyperpolarization makes the neuron more resistant to depolarization, causing depression of CNS function. The importance of this cellular mechanism in producing the clinical effects of barbiturates is unclear.

✔ GABA-mediated hyperpolarization of the neuron makes the neuron more resistant to depolarization, causing depression of CNS function.

Higher doses of barbiturates may directly activate the chloride ion channel independent of the barbiturate-GABA receptor complex (Levine, 1994). This results in a progressive depression of central nervous system function ranging from deep sedation to coma. When coma is induced, these agents are capable of decreasing cerebral metabolic oxygen consumption. Through autoregulation, the brain decreases cerebral blood flow, thereby lowering intracranial pressure.

Adverse Effects. These once widely used agents now have limited applications in critically ill patients because of dose-dependent cardiovascular and respiratory depression (Price-Roberts, 1984). Venodilation with peripheral pooling of blood, tachycardia, and depressed myocardial contractility may result in hypotension, especially in elderly patients with poor cardiac function.

✔ Venodilation with peripheral pooling of blood, tachycardia, and depressed myocardial contractility may result in hypotension in patients with poor cardiac function.

Respiratory adverse effects to barbiturates include blunted ventilatory response to hypoxia and hypercapnia, and reduced tidal volume and respiratory rate. Other adverse effects attributed to barbiturates include rashes and gastrointestinal upset.

Drug Interactions. Barbiturates may induce tolerance as well as physical and psychologic dependence. These concepts have been discussed at length in the section on opioid therapy. Another problem complicating barbiturate therapy is the potential for numerous drug interactions. When barbiturates are in use, liver metabolism of other drugs may be enhanced, thus decreasing their effects. Caution should be exercised when adding to or deleting a drug from a therapeutic regimen containing a barbiturate, giving consideration to the possible need for dosage adjustment.

✔ Barbiturates increase the clearance of those drugs that are metabolized by the liver.

Barbiturates do not relieve pain and may paradoxically heighten pain intensity (Dundee et al., 1988). Therefore it is necessary to reassess analgesic efficacy when these drugs are used in patients requiring analgesia.

✔ Barbiturates do not relieve pain and may paradoxically heighten the sensation of pain.

OTHER AGENTS USED IN MECHANICAL VENTILATION

Other agents used in mechanical ventilation include propofol (for sedation and maintenance of anesthesia), haloperidol (for treatment of delirium), dexmedetomidine (for sedation of mechanically ventilated patients), and nitric oxide (for dilation of pulmonary vessels).

Propofol

propofol (Diprivan): An intravenous drug that provides a spectrum of CNS effects ranging from light sedation to deep general anesthesia.

Propofol (Diprivan) is an intravenous (IV) drug administered together with other anesthetics to produce and maintain anesthesia. A hypnotic effect is produced within 40 sec after a rapid IV bolus administration (1 to 2.5 mg/kg). The maintenance dose is 0.05 to 0.2 mg/kg/min by IV infusion (Reiss et al., 2006). Propofol is used to provide sedation in ventilator patients and for induction and maintenance of anesthesia.

Mechanism of Action. Propofol appears to enhance GABA-activated chloride ion channel function (for a complete discussion, see Sedatives and Antianxiety Agents [Benzodiazepines] in this chapter) (Fragen et al., 1992). The mechanism of action may involve a separate receptor recognition site or may involve a different mechanism from that previously discussed for barbiturates and benzodiazepines.

Adverse Effects. Adverse reactions reported with propofol use include apnea, bradycardia, laryngospasm, bronchospasm, coughing, dyspnea, hypotension, and burning or pain at the site of infusion. Discoloration of the urine to green or red-brown may occur due to liver metabolites of propofol (Mirenda et al., 1995; Reiss et al., 2006).

✓ Propofol is formulated in an oil-in-water vehicle and it could contribute a significant amount of calories from fat.

Propofol is highly fat-soluble and is formulated in an oil-in-water vehicle. The soybean oil in this vehicle could contribute a significant amount of calories from fat. Therefore, other sources of dietary intake must be adjusted to compensate for the amount of fat infused. In addition, patients receiving propofol should be monitored for elevations in serum triglycerides. Because fat emulsion provides an excellent medium for microbial growth, and propofol contains no preservatives, strict aseptic administration techniques are important to prevent iatrogenic sepsis.

✓ Since fat emulsion provides an excellent medium for microbial growth, strict aseptic techniques are essential.

Clinical Considerations. Propofol has analgesic properties but only at doses used for general anesthesia, therefore attention must be paid to providing adequate analgesia in patients requiring pain relief. When opioid analgesics are used in combination with propofol, additive hypotension may occur.

✓ Propofol has analgesic properties but only at doses used for general anesthesia. Additional analgesics may be needed for pain control when used at sedative doses.

Since propofol does not promote salivation or vomiting, its use can be an advantage for intubated patients (Reiss et al., 2006). The rapid onset and offset of sedation may be a disadvantage if patients are allowed to awaken abruptly in pain or to great environmental disorientation (Levine, 1994). This problem can be avoided by decreasing the infusion rate so that the patient awakens slowly.

As discussed previously in the benzodiazepine section, monitoring the level of sedation is important to avoid over- or under-sedation. The Ramsay Scale (refer to Sedatives and Antianxiety Agents [Benzodiazepines] in this chapter for a discussion) or computer-assisted cerebral function monitoring can be used to determine the patient's level of sedation. The latter notes changes in brain function on a real-time basis and, with adequate training, the changes may be interpreted by caregivers.

Haloperidol

haloperidol (Haldol): A drug for the control of delirium in mechanically ventilated patients.

Pharmacotherapy for sedation and anxiolysis may in some patients paradoxically cause delirium. **Haloperidol (Haldol)** may be effective in these situations where control of delirium in mechanically ventilated patients is desirable. Delirium may be

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TABLE 13-21 Potential Procedures to Reverse Delirium

Withdrawal of Medications	Improvement of Clinical Conditions
Alcohol	Fever
Analgesics	Head injury
Anticholinergics	Hepatic failure
Anticonvulsants	Hyperparathyroidism and hypoparathyroidism
Antihistamines	
Antihypertensives	Hyperthyroidism and hypothyroidism
Antiparkinsonian agents	Renal failure
Cardiac drugs	
Corticosteroids	
Psychiatric agents	

Haloperidol (Haldol) may be effective in sedating patients who have concurrent agitation and delirium caused by use of sedatives.

defined as a reversible global impairment of cognitive processes manifested chiefly by disorientation, impaired short-term memory, arousal, attention, illusions, and hallucinations (McCartney et al., 1993). The initial approach to a ventilator patient presenting with delirium should be a search for reversible causes. See Table 13-21 for potential procedures to reverse delirium.

Nonpharmacologic approaches such as repeated reorientation and explanation (explain treatment plans to the patient) minimize environmental stress and enhance communication (written or hand signals in intubated patients may also be helpful). Drug therapy should be added to nonpharmacologic approaches if the latter are ineffective and a reversible cause is not found.

Indications. Haloperidol is primarily used in the critically ill, mechanically ventilated patient for the control of delirium. The intravenous route of administration is preferred in the intensive care setting, although the drug may be given intramuscularly or orally.

Mechanism of Action. An increase in dopamine release and metabolism have been postulated as one of the central nervous system derangements manifesting as delirium. Haloperidol blocks dopamine receptors in the central nervous system (limbic, basal ganglia, and brain stem), producing a calming effect (McEvoy, 1995). In addition, haloperidol blocks dopamine receptors in the chemoreceptor trigger zone that may be responsible for its **antiemetic** activity.

antiemetic: Preventing nausea and vomiting.

Adverse Effects. Blockade of dopamine receptors in the central nervous system may interfere with normal motor function. These adverse effects are called extrapyramidal

✓ Neck twisting, swollen tongue, jaw muscle spasm, and flexion of head and feet backward are some adverse effects of haloperidol.

reactions (EPS) and may include unilateral cervical muscle contraction with neck twisting (torticollis), swollen tongue (laryngeal dystonia), jaw muscle spasm (trismus), and flexion of head and feet backward (opisthotonus) (McEvoy, 1995). EPS occurs much less frequently with IV haloperidol than that observed with intramuscular or oral therapy. The reason for this is currently unknown (Fish, 1991).

Neuroleptic malignant syndrome is a rare, idiosyncratic, life-threatening reaction that may occur after a single dose of haloperidol. Hyperthermia, altered consciousness, labile blood pressure, diaphoresis, and tachyarrhythmias are suggestive of this condition (Simon, 1993).

Haloperidol may also prolong the electrocardiographic QT interval that on rare occasions can produce a polymorphic form of ventricular tachycardia known as torsade de pointes (Fish, 1991).

✓ Combined use of a benzodiazepine, opioid, and haloperidol is often necessary for control of extremely agitated, delirious patients.

Clinical Considerations. Combination therapy including a benzodiazepine, opioid, and haloperidol is often necessary for control of extremely agitated, delirious patients requiring critical care. The critical care team must be diligent in the search for reversible causes of delirium and, if found, must correct them whenever possible.

Dexmedetomidine

dexmedetomidine (Precedex): An intravenous drug that offers anxiolysis and analgesia but no respiratory depression.

For decades, gamma-aminobutyric acid (GABA) receptor agonists (e.g., propofol, midazolam) have been used extensively as a sedative of choice in the intensive care units (Riker et al., 2009). **Dexmedetomidine (Precedex)** is a newer intravenous drug (since 1999 in the U.S.) that offers anxiolysis and analgesia but no respiratory depression (Bekker et al., 2005). The lack of respiratory depression is desirable for the management of mechanically ventilated patients, especially during measurement of weaning mechanics and evaluation of weaning feasibility.

Indications. Dexmedetomidine is indicated for sedation in mechanically ventilated patients. It can be used as a continuous infusion prior to extubation, during extubation, and postextubation. It is not necessary to discontinue dexmedetomidine prior to extubation. Dexmedetomidine is also used for sedation of patients prior to and during cardiac or vascular surgeries or other uncomfortable procedures such as colonoscopy (Precedex, 2012). Intranasal dexemedetomidine is another route of administration for children undergoing MRI and CT procedures. Since this drug has a neutral pH, it is painless when given intranasally (Phillips, 2010).

✓ Dexmedetomidine (Precedex) provides sedation and anxiolysis via receptors within the locus ceruleus, analgesia via receptors in the spinal cord, and gradual reduction of stress response with no significant respiratory depression.

Mechanism of Action. Dexmedetomidine is an α_2 adrenoreceptor agonist with a unique combination of physiologic actions. It provides sedation and anxiolysis via receptors within the locus ceruleus (group of neurons in the pons), analgesia via receptors in the spinal cord, and gradual reduction of stress response with no significant respiratory depression (Riker et al., 2009).

Adverse Effects. Dexmedetomidine should be administered using a dosage-controlled infusion device, and the manufacturer recommends a duration of infusion not to exceed 24 hours. However, randomized clinical trials comparing the drug to midazolam and lorazepam have demonstrated efficacy and safety for up to 5 days of continuous use (Riker et al., 2009). Because dexmedetomidine decreases sympathetic

✓ Adverse reactions of dexmedetomidine (Precedex) include hypotension, bradycardia, and sinus arrest.

nervous system activity, clinically significant episodes of hypotension, bradycardia, and sinus arrest are potential adverse reactions of this drug. Hypotension or bradycardia may be more pronounced in patients with hypovolemia, diabetes mellitus, chronic hypertension, and in elderly patients. On occasion, transient hypertension may develop. For these reasons, cautions should be exercised when administering this drug to patients with advanced heart block or severe left ventricular dysfunction (Precedex, 2012). In some children, transient neurological abnormalities (i.e., decreased verbal communication, facial drooping, and unilateral pupil dilation) have been observed upon discontinuation of the medication (Honey et al., 2010).

Clinical Considerations. Due to the known pharmacological effects of dexmedetomidine, patients receiving this drug should be monitored continuously for signs of cardiovascular instability. If persistent or severe hypotension and bradycardia occur, the infusion should be decreased or discontinued. Mild hypotension from this drug may be managed by increasing the rate of intravenous fluid, elevating the lower extremities, and using vasopressors (rtlist, 2012).

Nitric Oxide

nitric oxide: Inhaled nitric oxide (iNO) therapy has been used to treat persistent pulmonary hypertension and hypoxemic respiratory failure of the newborn, respiratory distress syndrome and hypoxemic respiratory failure of older infants and children, and acute respiratory distress syndrome in adults.

Nitric oxide is endogenously synthesized in vascular endothelium from the amino acid L-arginine (Palmer et al., 1987). After being released from the endothelium, nitric oxide diffuses into vascular smooth muscle cells where it stimulates production of 3′5′-cyclic guanosine monophosphate (cGMP). cGMP decreases the concentration of calcium in the muscle, resulting in vasodilatation. Because nitric oxide is extremely labile (rapidly inactivated by hemoglobin) it causes only local regulation of endothelial tone without systemic hypotensive effects.

Indications. Inhaled nitric oxide (iNO) therapy has been used as a treatment or supportive modality for a variety of clinical conditions. Some of these conditions include persistent pulmonary hypertension and hypoxemic respiratory failure of the newborn (Abman & Kinsella, 1999), respiratory distress syndrome and hypoxemic respiratory failure of older infants and children (Dobyns et al., 1999), and acute respiratory distress syndrome (Burke-Martindale, 1998). In addition, iNO therapy has been effective in increasing pulmonary blood flow and oxygenation and improving systemic cardiopulmonary hemodynamics in infants (Ochikubo et al., 1997).

✓ Inhaled nitric oxide produces local vasodilation of vascular smooth muscles.

✓ Pulmonary vasodilation may reduce pulmonary vascular resistance, correct V/Q mismatch, and improve oxygenation.

Mechanism of Action. Inhaled nitric oxide produces local vasodilation of vascular smooth muscles. Because the gas is delivered to alveolar units still presumably participating in gas exchange, these alveolar capillary units are preferentially vasodilated, diverting blood from hypoxic capillary beds with high vascular resistance (Stamler et al., 1992). The net physiologic results are reduction of pulmonary vascular resistance, reversal of hypoxic pulmonary vasoconstriction in unobstructed airway (improvement of V/Q matching), and improvement of oxygenation (Figure 13-8).

Adverse Effects. Adverse effects of iNO therapy are dependent on the dosage and concentration of inhaled NO. When combined with oxygen, NO is converted to nitrogen dioxide (NO_2). NO_2 levels of higher than 10 ppm can cause cell damage, hemorrhage, pulmonary edema, and death. However, at therapeutic dosages

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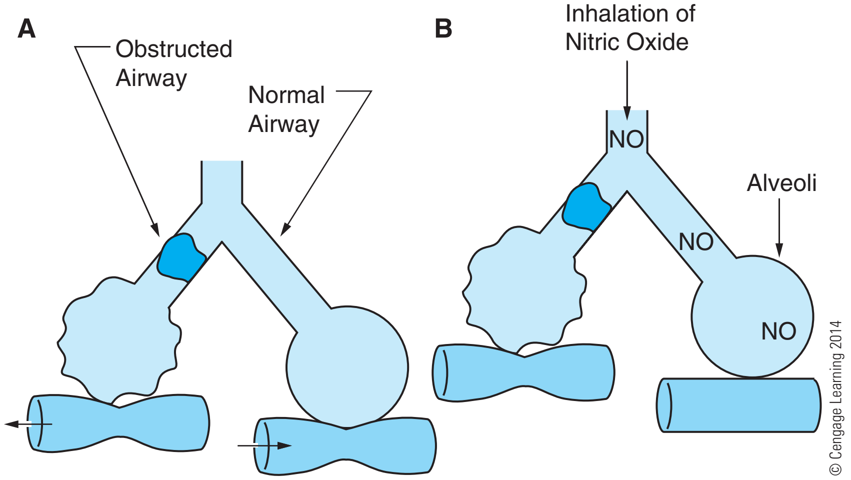

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FIGURE 13-8 (A) Ventilation/perfusion (V/Q) relationship of an obstructed airway and a normal airway. The perfusion to both is decreased because of hypoxic vasoconstriction. In the obstructed airway, V/Q is absent. In the unobstructed airway, a high V/Q (deadspace ventilation) is seen. (B) Inhalation of nitric oxide (NO) selectively dilates the pulmonary vessels of the ventilated alveoli, thus improving perfusion and restoring normal V/Q in unobstructed airway.

(less than 2 ppm), adverse effects and toxicity caused by inhalation of NO are rare (Linberg & Rydgren, 1999; Wilkins et al., 2008).

✓ Nitric acid and nitrous acid are lung inflammatory by-products of nitric oxide therapy.

NO and NO_2 may be converted to nitric acid (HNO_3) and nitrous acid (HNO_2), respectively, both of which may cause lung inflammation (interstitial pneumonitis). Toxicology information on these compounds is primarily available from administration of various concentrations of NO and NO_2 to healthy animals or volunteers with normal lungs (Lunn, 1995). Further research is necessary to determine the adverse effects of prolonged exposure of NO and NO_2 in patients with lung diseases.

NO is inactivated by combining with hemoglobin to form methemoglobin (hemoglobin in blood oxidized to ferric form that is incapable of transporting oxygen). The amount formed during therapy depends on the amount of NO administered and the amount of methemoglobin eliminated by an enzyme (methemoglobin reductase). Partial or complete deficiencies of this enzyme may exist in some patients, predisposing them to methemoglobinemia. The signs and symptoms of methemoglobinemia are listed in Table 13-22 (Dabney et al., 1990).

Other potential adverse effects of NO are inhibition of platelet aggregation and possible negative inotropic effect.

Clinical Considerations. Most of the patients who receive iNO therapy are on mechanical ventilation. Two components are necessary for the delivery of NO to these patients: inline monitoring of NO and NO_2, and a delivery source of NO gas (Wessel et al., 1994).

At this time, NO is regulated by the U.S. Food and Drug Administration (FDA) and is available as an investigational agent. It is supplied as cylinders containing either 800 parts per million (ppm) or 2,200 ppm with nitrogen as the inert balance gas.

Delivery System and Dosage. INOvent (INO Therapeutics, Inc. for North America and Europe; Datex-Ohmeda for elsewhere) is a U.S. FDA-approved delivery system for

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TABLE 13-22 Signs and Symptoms of Methemoglobinemia

Methemoglobin Concentration	Clinical Signs and Symptoms
10% to 20%	Central cyanosis of trunk and limbs, usually asymptomatic
20% to 45%	CNS depression (headache, dizziness, fatigue, lethargy, syncope)
45% to 55%	Coma, arrhythmia, shock, convulsion
>70%	High risk of death

inhaled nitric oxide therapy. It works with a ventilator or anesthesia machine to deliver a precise and stable concentration of nitric oxide, regardless of the ventilator setting or mode. Alarms are built-in to ensure safe and reliable therapy with continuous monitoring of NO, NO_2, and O_2. The INOvent design also integrates with a system for delivering nitric oxide via a manual resuscitator. For NO therapy guidelines, refer to the document by Klein (2012) in the Additional Resources section at the end of the chapter.

SUMMARY

In mechanical ventilation, different drugs are used to (1) provide bronchodilation, (2) facilitate intubation, (3) relieve pain and anxiety, (4) treat seizures or elevated intracranial pressure, and (5) maintain patient-ventilator synchrony. These drugs can be very useful when they are used as directed. At the same time, they must be used with care, since adverse effects can be dangerous or even life-threathening. Practitioners are expected to have a thorough knowledge of the indications, contraindications, and adverse effects of the drugs that are administered to patients on mechanical ventilation.

Self-Assessment Questions

1. Bronchodilation may be induced by all of the following mechanisms *except*:
   - A. stimulating the sympathetic branch of the autonomic nervous system.
   - B. stimulating the parasympathetic branch of the autonomic nervous system.
   - C. inhibiting the enzyme phosphodiesterase.
   - D. inhibiting the parasympathetic branch of the autonomic nervous system.

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2. A patient who has been using Theo-Dur at home states that she experiences palpitations, nausea, vomiting, headache, and agitation each time after taking the medication. The proper management technique for this patient includes monitoring of the _______ level and titrating the dosage to a therapeutic range of _______.

   A. serum theophylline, 5–15 mcg/mL
   B. corticosteroid, 5–15 mcg/mL
   C. serum theophylline, 40–60 mcg/mL
   D. corticosteroid, 40–60 mcg/mL

3. An asthmatic patient who has been using Ventolin for several months complains of shortness of breath and states that "the Ventolin is not working any more." The physician asks a therapist for suggestion. The therapist should recommend a trial use of:

   A. Proventil.
   B. Alupent.
   C. Brethine.
   D. Vanceril.

4. Which of the following is *not* an indication for using a neuromuscular blocking agent?

   A. relieve laryngeal spasm
   B. maintain mechanical ventilation
   C. facilitate endotracheal suctioning
   D. enhance endotracheal intubation

5. Succinylcholine (Anectine, Quelicin) is a _______ neuromuscular blocking agent. It induces muscle blockade by _______.

   A. depolarizing, binding to the receptor sites and causing sustained depolarization
   B. nondepolarizing, binding to the receptor sites and causing sustained depolarization
   C. depolarizing, competing for the receptor sites and blocking the action of acetylcholine
   D. nondepolarizing, competing for the receptor sites and blocking the action of acetylcholine

6. All of the following are potential complications of a neuromuscular blocking agent *except*:

   A. oversedation.
   B. prolonged muscle blockade.
   C. apnea.
   D. bronchospasm.

7. A patient who has been given Norcuron one hour ago is recovering in the surgical intensive care unit. The physician wants to know whether neuromuscular blockade reversal has occurred. The therapist should check all of the following *except*:

   A. tongue protusion.
   B. serum acetylcholine level.
   C. hand grip.
   D. head lift.

8. A mechanically ventilated patient has been receiving diazepam (Valium) for better tolerance of mechanical ventilation and related procedures. The cardiovascular adverse effects for his patient may include:

   A. decreased cardiac output.
   B. increased stroke volume.
   C. increased mean arterial pressure.
   D. increased systemic vascular resistance.

9. The Ramsay Scale is commonly used to assess the degree of:

   A. ventilation.
   B. pain.
   C. bronchodilation.
   D. sedation.

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10. Inadequate pain control may lead to all of the following complications *except*:

    A. increased blood pressure.
    B. increased immune function.
    C. decrease of lung function.
    D. decrease of patient mobility.

11. A patient is given naloxone upon returning from surgery. You may conclude that the patient has received too much _______ during surgery.

    A. opioid analgesic
    B. neuromuscular blocking agent
    C. benzodiazepine
    D. nitric oxide

12. For seizure disorders and control of elevated intracranial pressure, _______ may be useful.

    A. neuromuscular blocking agent
    B. nitric oxide
    C. barbiturate
    D. corticosteroid

13. Haloperidol (Haldol) should be considered for controlling delirium:

    A. as soon as a patient is placed on a mechanical ventilator.
    B. when narcotic analgesic is given to a patient.
    C. when a patient experiences pain.
    D. after reversible causes of delirium have been ruled out.

14. Dexmedetimidine is a GABA agonist that provides all of the following pharmacological effects *except*:

    A. sedation.
    B. anxiolysis.
    C. analgesia.
    D. respiratory stimulation.

15. Among other applications, inhaled nitric oxide (iNO) is used to treat _______. When iNO is combined with oxygen, _______ is formed.

    A. pulmonary hypotension, nitrogen dioxide
    B. pulmonary hypotension, nitrogen trioxide
    C. pulmonary hypertension, nitrogen dioxide
    D. pulmonary hypertension, nitrogen trioxide

Answers to Self-Assessment Questions

1. B.
2. A.
3. D.
4. C.
5. A.
6. A.
7. B.
8. A.
9. D.
10. B.
11. A.
12. C.
13. D.
14. D.
15. C.

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Chapter 14

Procedures Related to Mechanical Ventilation

David W. Chang

Outline

Introduction
Chest Tube and Drainage System
  Indications for Chest Tube
  Chest Tube Selection and Placement
  Methods of Placement
  Chest Tube Drainage System
  Care and Removal of Chest Tube
  Transport with Chest Tube
Assisting in Fiberoptic Bronchoscopy
  Indications for Fiberoptic Bronchoscopy
  Bronchoscope and Medications
  Insertion of Bronchoscope
  Type of Specimen
  Complications
  Postbronchoscopy Care
Transport of Mechanically Ventilated Patients
  Indications
  Contraindications
  Equipment and Supplies
  Types of Transport
  Procedures for Interhospital Transport
  Hazards and Complications
  Magnetic Resonance Imaging
Summary
Self-Assessment Questions
Answers to Self-Assessment Questions
References
Additional Resources

Key Terms

atropine sulfate
bronchial brushing
chest tube (thoracostomy tube)
fiberoptic bronchoscope
forceps biopsy
interhospital transport

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intrahospital transport
lidocaine
operative tube thoracostomy
three-chamber drainage system
transbronchial lung biopsy (TBLB)
transbronchial needle aspiration biopsy (TBNAB)
transport ventilator
trocar
trocar tube thoracostomy

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Describe the clinical application, setup, and discontinuance of a three-column chest tube and drainage system.
- Describe the indications and medications for fiberoptic bronchoscopy.
- List the type of specimen obtained from a fiberoptic bronchoscopy procedure.
- List the complications of fiberoptic bronchoscopy.
- List the indications, contraindications, equipment, and supplies for transport of a mechanically ventilated patient.
- Describe the procedures for intrahospital and interhospital transport of a mechanically ventilated patient.

INTRODUCTION

This chapter covers three clinical topics related to mechanical ventilation: chest tube and drainage system, assisting in fiberoptic bronchoscopy, and transport of mechanically ventilated patients. A sound conceptual foundation and working knowledge of the equipment and supplies for these procedures can ensure patient safety and enhance patient care.

CHEST TUBE AND DRAINAGE SYSTEM

chest tube (thoracostomy tube): A tube that connects the pleural space and drainage system for removal of air or fluid.

Chest tube (thoracostomy tube) and vacuum drainage system are commonly used in acute care settings on patients breathing spontaneously or receiving mechanical ventilation. Respiratory care practitioners should have a good working knowledge of these devices so that hazards and complications with the chest tube setup may be recognized and corrected without delay.

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Indications for Chest Tube

Common indications for chest tube include large pneumothorax (>25%), hemothorax, and pleural effusion.

Common indications for chest tube include large pneumothorax (>25%), hemothorax, and pleural effusion. Causes of pneumothorax include positive pressure ventilation, ruptured bleb due to emphysema, bronchopleural fistula, leaking subpleural cyst, and chest trauma. Iatrogenic pneumothorax may be caused by invasive procedures such as thoracentesis, central vein/pulmonary artery catheterization, and bronchoscopy/transbronchial biopsy (Chen et al., 2002). Excessive pleural fluid may be caused by thoracic trauma (hemothorax), heart failure (pleural effusion), intra-abdominal infection (empyema), and blockage of lymphatic system (chylothorax) (Irwin et al., 2003).

Potential complications of chest tube placement include hemorrhage at insertion site, hematoma, laceration of lung parenchyma or intra-abdominal organs, and infection.

Contraindications and Complications. There are no absolute contraindications to chest tube placement in patients who are symptomatic from the above indications. Relative contraindications include infection over the insertion site, and conditions that may lead to severe bleeding during chest tube placement (Thomas, 2004).

Potential complications of chest tube placement include hemorrhage at the insertion site, hematoma, and laceration of lung parenchyma or intra-abdominal organs. Infection can be a late complication (Thomas, 2004).

The suggested chest tube size for teens and large adults ranges from 28 to 40 Fr.

Chest Tube Selection and Placement

trocar: A trocar is a sharply pointed instrument for incision into the chest cavity.

The chest tube may be straight, curved, trocar, or nontrocar. A **trocar** is a sharply pointed instrument for incision into the chest cavity (Figure 14-1). The suggested chest tube size for large adults ranges from 36 to 40 French (Fr). For small adults or teens, a range from 28 to 32 Fr is adequate. For children and neonates, the appropriate chest tube sizes are 18 and 12 to 14 Fr, respectively.

Since clotting of the chest tube with blood or pleural fluid is less likely in pneumothoraces, 16 to 20 Fr may be adequate for adults.

Clotting of blood inside the chest tube is a potential complication following placement. For this reason, larger tubes are selected for treatment of hemothorax or other thick pleural fluids (e.g., empyema). Since clotting is less likely when the chest tube is used to treat pneumothoraces, smaller sizes (16 to 20 Fr) may be adequate for adults.

To treat pneumothorax, the chest tube (16 to 20 Fr) is placed at the second or third intercostal space anteriorly along the midclavicular line or midaxillary line.

To drain fluid, the chest tube is placed from the fourth to sixth intercostal space at the midaxillary line.

Chest Tube Placement. Chest tubes are inserted under sterile conditions at the bedside or in surgery. To treat pneumothorax, the chest tube (16 to 20 Fr) is usually placed at the second or third intercostal space anteriorly along the midclavicular line or midaxillary line. For drainage of blood or other pleural fluids, a size 36 Fr (ranging from 28 to 40 Fr) chest tube is recommended to reduce clotting. The insertion point is from the fourth to sixth intercostal space at the midaxillary line (usually a line lateral to the nipple) for optimal drainage of pleural fluid (Figure 14-2).

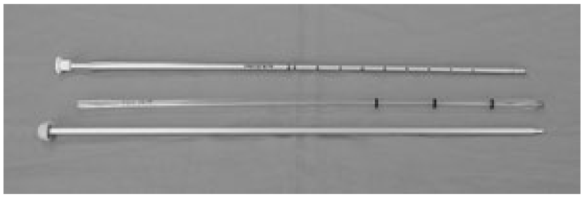

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FIGURE 14-1 A chest tube with trocar (lower solid piece). The trocar is placed inside the chest tube for insertion through the incision on the chest wall.

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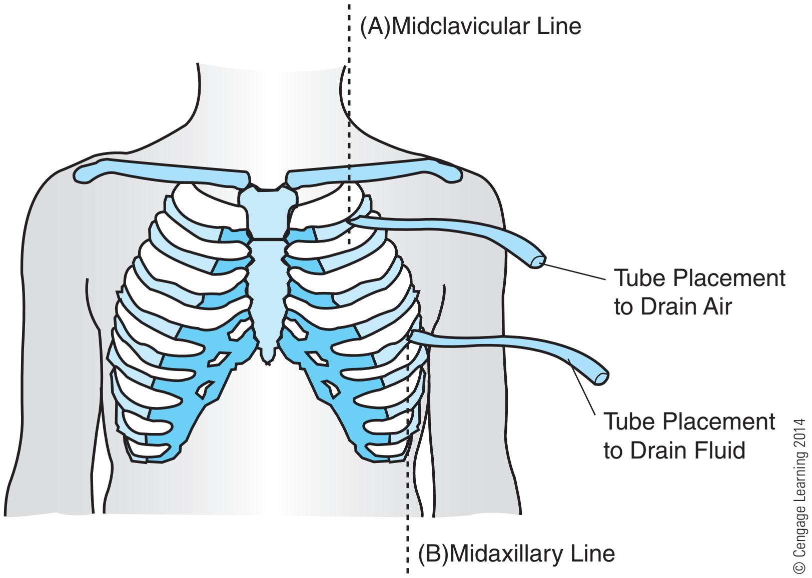
(A)Midclavicular Line
Tube Placement to Drain Air
Tube Placement to Drain Fluid
(B)Midaxillary Line
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FIGURE 14-2 Chest tube insertion sites showing (A) second intercostal space anteriorly along the midclavicular line for relieve of pneumothorax; (B) fifth intercostal space along the midaxillary line for drainage of pleural fluid.

✔ The point of entry is directly over the body of the rib because arteries, veins, and intercostal nerves all lie below each rib.

During chest tube placement, the point of entry is directly over the body of the rib to avoid complications. Incisions or punctures are done above the rib because arteries, veins, and intercostal nerves all lie below each rib. Figure 14-3 shows the correct placement of a chest tube entering the chest wall at the midaxillary line. The point of entry is directly over the rib.

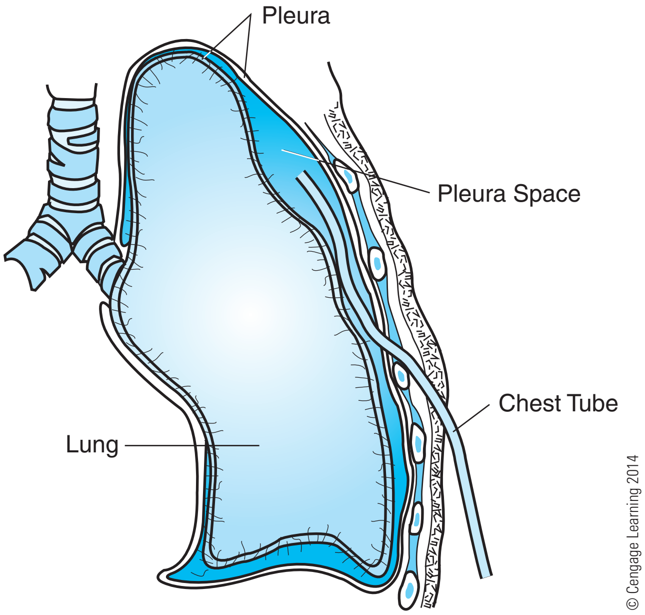
Pleura
Pleura Space
Chest Tube
Lung
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FIGURE 14-3 Correct placement of a chest tube entering the chest wall at the midaxillary line. The point of entry is directly over the rib to avoid puncture of the arteries, veins, and intercostal nerves that lie below each rib.

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Methods of Placement

Operative tube thoracostomy and trocar tube thoracostomy are two common methods to perform chest tube placement. Each method has its advantages and disadvantages (Deshpande et al., 2002).

operative tube thoracostomy: A technique of chest tube placement by dissection into the pleura, digital inspection of the pleural space, and insertion guided with the finger and hemostat.

Operative Tube Thoracostomy. In **operative tube thoracostomy**, the incision is made parallel to and above the rib. It is followed by blunt dissection into the pleura. A finger is inserted into the opening for inspection of the pleural space. A chest tube is then guided into the pleural space by using a finger and hemostat or Kelly clamp (Figure 14-4). This method is safer than trocar tube thoracostomy because digital inspection eliminates the possibility of chest tube placement between the parietal pleura and the chest wall. However, it is more involved and requires a larger incision to allow the finger, chest tube, and hemostat to enter the chest wall and the pleural space.

trocar tube thoracostomy: A technique of chest tube placement by incision into the pleura, insertion of trocar chest tube, and withdrawal of trocar.

Trocar Tube Thoracostomy. In **trocar tube thoracostomy**, the incision is also made parallel to and above the rib. The chest tube with trocar inside is inserted through the incision (Figure 14-5). The chest tube/trocar setup should enter the chest only 1 to 2 cm, otherwise puncture of the lung is likely. Once inside the pleural space, the chest tube is advanced over the trocar—a procedure similar to the "catheter over needle" technique for artery line placement. The chest tube is clamped with a forceps before complete withdrawal of the trocar. This method requires a smaller incision and provides less tissue trauma and less patient discomfort.

Following placement, the rigid chest tube is connected to the flexible Creech tubing with a clear, ridged plastic connector flange. Since the flange has a narrow diameter, any clots from the pleural cavity may become lodged at this location. When cloth tape is used to seal and secure the connection, it should be done in a way that does not interfere with the visual inspection of any clot formation inside the connector.

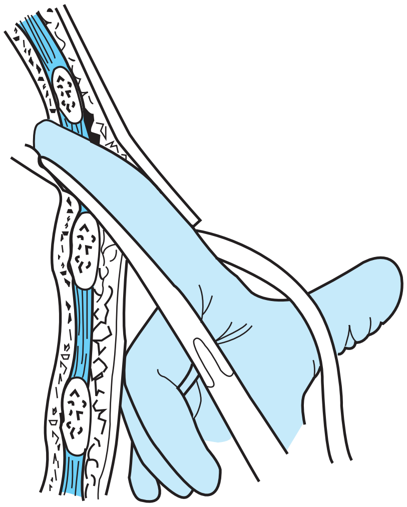

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FIGURE 14-4 The chest tube is clamped by a hemostat and both are guided into the pleural space by a finger.

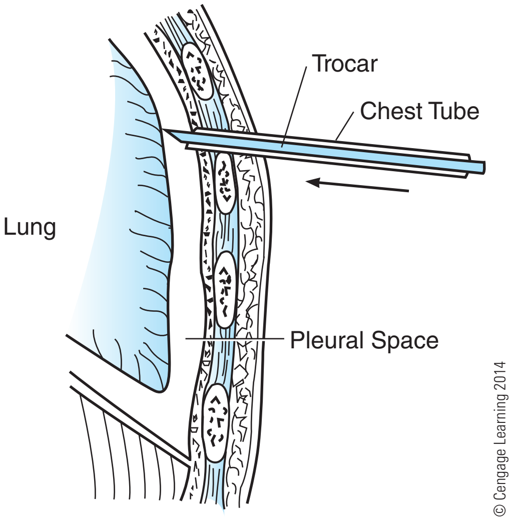

FIGURE 14-5 Incision is done parallel to and above the rib. The chest tube with trocar inside is inserted through the incision over the rib.

✓ Kinking of the chest tube will lower the suction level, hinder lung re-expansion, and cause the fluid to enter the pleural space.

The chest tube should be kept in a straight line as much as possible. If the tube is looped or kinked, the suction level will decrease and lung re-expansion may be hindered. In addition, fluid in the tube may re-enter the pleural space, leading to the possibility of infection.

Chest Tube Drainage System

Following insertion and inspection of the chest tube for proper placement, the chest tube is immediately connected to a drainage system that has been previously set up. Since fluid is gravity-dependent, all chest tube drainage systems are placed below the level of chest tube placement. There are different drainage systems for a chest tube. The most common and versatile drainage system in the hospital is the three-chamber setup such as Pleur-Evac®. Proper functioning of the drainage system must be evaluated, and unusual occurrence with the drainage system must be correlated to the patient's condition and vital signs.

The one-chamber and two-chamber drainage systems are simple in design and they can be set up quickly. While these two systems do not require a vacuum source, their usefulness in intensive care settings is rather limited. The **three-chamber drainage system** is the most versatile one and it requires a vacuum source to provide continuous suction. All three systems are discussed below for a clear concept of the working mechanism of a chest tube drainage system.

three-chamber drainage system: A chest tube drainage setup that requires a vacuum source to provide continuous suction.

One-Chamber System. In a one-chamber water-seal system (Figure 14-6A), the chamber initially contains 100 mL of sterile water. The short air-vent tube prevents pressure buildup in the chamber. The long tube is submerged in exactly 2 cm of water. This acts as a water seal which allows air to escape but not to return. With this system, the work of spontaneous breathing is proportional to the submersion depth of the long tube. As pleural fluid drains and accumulates in the chamber, more of the long tube is submerged and spontaneous breathing becomes more difficult.

✓ As pleural fluid drains and accumulates in the one-chamber system, more of the long tube is submerged and spontaneous breathing becomes more difficult.

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Some bubbling from the long tube is normal as long as there is air in the pleural space. If there is no bubbling, either there is an obstruction or there is no more air in the pleural space.

✓ In a two-chamber drainage system, chamber 1 collects all pleural fluids. The fluid level in chamber 2 remains constant and the work of spontaneous breathing is unaffected.

Two-Chamber System. The two-chamber drainage system (Figure 14-6B) uses chamber 1 to collect drainage or pleural fluid or evacuate pleural air. The amount of pleural drainage can be measured from chamber 1. In chamber 2, the short tube allows air to escape and prevents pressure to build up. The long tube in chamber 2 is submerged in 2 cm of water as in the one-chamber drainage system. Since chamber 1 collects all pleural fluid, the submersion depth of the long tube in chamber 2 remains unchanged. The work of spontaneous breathing is therefore unaffected by the volume of pleural fluid collected in this chamber.

Three-Chamber System. The three-chamber drainage system is the most common chest tube drainage system (Figure 14-6C). Chamber 1 (collection chamber) collects the pleural fluid from the patient. Chamber 2 (water seal chamber) has about

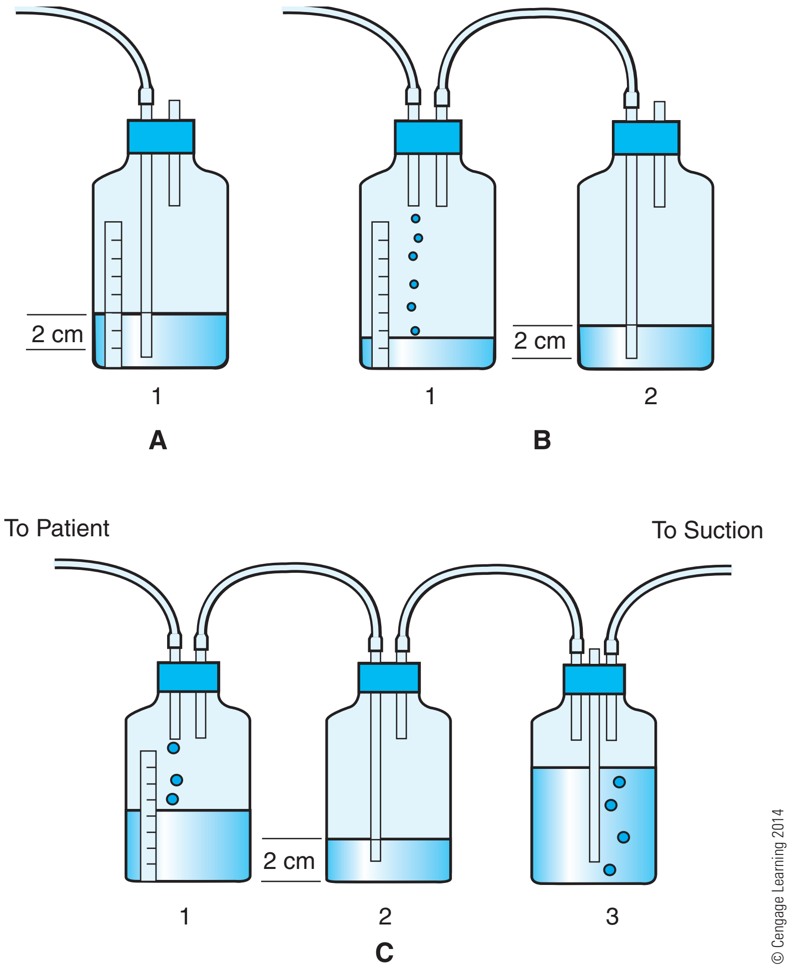

FIGURE 14-6 Chest tube drainage and water seal systems. (A) One-chamber system; (B) Two-chamber system; (C) Three-chamber system. The amount of bubbling is controlled by the vacuum source and it does not indicate the level of suction applied to the pleural space. The level of suction applied to the pleural space is determined by the submersion depth of the vent tube in the suction chamber.

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✔ The water level in chamber 3 (suction chamber) regulates the amount of suction in the three-chamber system.

✔ The setting of wall vacuum and the amount of bubbling in chamber 3 do not reflect the level of suction applied to the pleural space.

2 cm of water in it and functions as a water seal. The water level in chamber 3 (suction chamber) regulates the amount of suction in the three-chamber system. For example, a suction level of −15 cm H_2O can be achieved by adding sterile water into this chamber to a height of 15 cm H_2O. A low suction level (−10 to −20 cm H_2O) is recommended for the chest tube drainage system.

Under normal working condition, the vacuum draws air into the fluid through the venting tube in chamber 3, causing a constant slow bubbling effect. Too much bubbling means the vacuum level is set too high. The setting of wall vacuum and the amount of bubbling do not reflect the level of suction applied to the pleural space. The level of suction applied to the pleural space is determined by the submersion depth of the venting tube in suction chamber 3. For this reason, the water level in suction chamber 3 must be monitored and kept at the appropriate level in order to maintain a desired vacuum level (from −10 to −20 cm H_2O). Evaporative water loss will lower the submersion depth and decrease the suction level.

The one- and two-column drainage systems drain fluid by gravity. If suction is desired, a three-column system must be used. Figure 14-7 shows a typical chest tube drainage system that combines all three chambers in one unit.

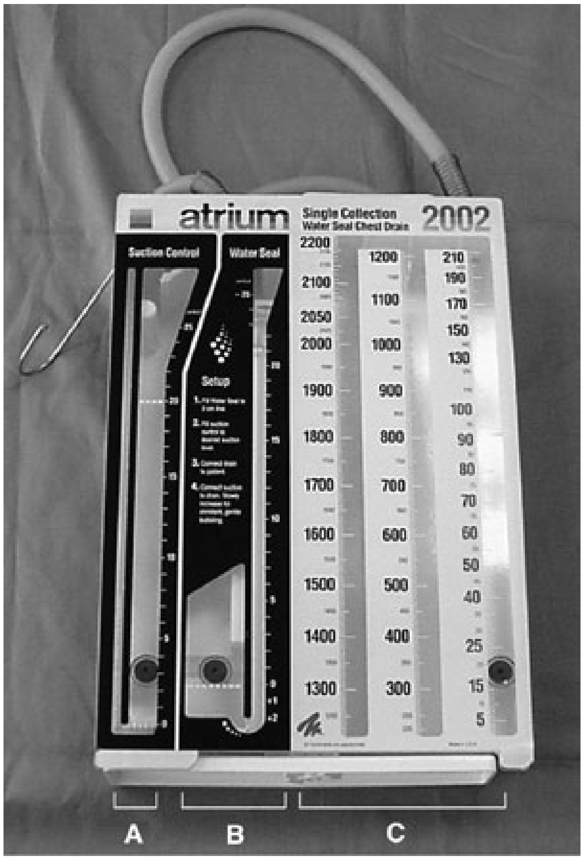

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FIGURE 14-7 A typical chest tube drainage system that combines all three columns [suction control (A), water seal (B), and collection (C)] in one unit. In some drainage systems, the cap for the water seal column has a pressure relief valve which is closed under normal suction and opens in case of excessive pressure caused by blocked suction source or obstructed suction tubing. This feature prevents excessive pressure being transmitted to the chest tube and pleural space.

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With a three-chamber drainage system, the fluid collection chamber should be inspected to note the volume and characteristics of the fluid drainage. The volume collected should decrease over time.

Care and Removal of Chest Tube

✓ If the drainage holes on the chest tube become visible, the chest tube has come out too far.

✓ If the chest tube becomes disconnected from the patient, an occlusive dressing such as Vaseline gauze must be applied immediately over the incision opening.

✓ If a large amount of bubbling is observed in water seal chamber 2, air leak in the drainage system or presence of air in the pleural space may be the cause.

✓ Overfilling of water in suction chamber 3 will increase the suction level to the pleural space, whereas low water level will reduce the suction level.

Emergencies may happen to the chest tube setup. If the drainage holes on the chest tube become visible, the physician should be notified immediately for repositioning or reinsertion. If the chest tube becomes disconnected from the patient, an occlusive dressing such as Vaseline gauze should immediately be applied over the incision opening. The physician is then notified and the patient should be monitored closely for signs of respiratory distress. If the chest tube is disconnected from the drainage unit, clamp the chest tube and reconnect it with a new drainage unit. Clamping of the chest tube should not exceed 1 min.

The water level in the middle (water seal) chamber normally fluctuates with respiration. This means the tube and drainage system are working properly. If a large amount of bubbling is observed in the middle chamber, air leak in the drainage system or presence of air in the pleural space may be the cause. The patient, connections, vacuum level, and amount of sterile water in the drainage unit should be checked for the source of air leaks.

In order to maintain a desired suction level between −10 and −20 cm H_2O, the water level in the suction chamber must be kept at the appropriate level by filling it with sterile water as needed. Overfilling of water in this chamber will increase the suction level to the pleural space, whereas low water level will reduce the suction level.

The chest tube can be removed when the pleural drainage has stopped or slowed to less than 100 mL over the preceding 24 hours, or when the pneumothorax has resolved and there is no further air leak. Air leak (bubbling in the middle chamber) may be tested by asking the patient to perform a Valsalva's maneuver or a forceful cough (Alameda County Medical Center, 2004).

The suture is first removed and the patient is instructed to perform a Valsalva's maneuver right before pulling out the chest tube. A petrolatum gauze and dressing are applied to the opening immediately. Follow-up chest radiography is done in 4 hours to allow proper lung re-expansion and to detect reoccurring pneumothorax (Alameda County Medical Center, 2004).

Transport with Chest Tube

On occasion, patients with a chest tube setup may need to go to another location for testing or treatment. In addition to an oxygen source, primary emergency drugs and airway equipment should be available during transport. The transport team must properly maintain the chest tube and drainage system during the entire transport process. The drainage system must be lower than the patient's chest at all times. The chest tube must be functional and the patient's pretransport vital signs must be monitored and maintained to ensure stable patient condition.

TABLE 14-1 Transport with Chest Tube

1. The drainage system must be kept lower than the patient's chest at all times.
2. The chest tube must not be clamped or occluded at any time.
3. The chest tube must be connected to a functional water seal drainage system.
4. The drainage system must not be tipped or dropped.

✓ During transport, the drainage system must be kept lower than the patient's chest and the chest tube must not be clamped or occluded.

The chest tube must never be clamped or occluded, because this action may affect mechanical or spontaneous ventilation and reverse the re-expansion of the affected lung. Furthermore, fluid flowing back into the pleural space is a source of infection. In most severe cases, a clamped chest tube may potentially convert a simple pleural air leak to life-threatening tension pneumothorax. Therefore, the chest tube must be unobstructed and connected to a functional water seal drainage system. The water seal allows air or fluid to exit the pleural space and prevents it being drawn back into the pleural space (Jacobsohn, 2004). Table 14-1 summarizes the key points to ensure proper functioning of a chest tube during patient transport.

ASSISTING IN FIBEROPTIC BRONCHOSCOPY

fiberoptic bronchoscope: An instrument that uses glass fibers to transmit images of the airway for diagnostic or therapeutic procedures under direct vision.

A **fiberoptic bronchoscope** is used to provide diagnostic and therapeutic procedures for conditions involving the airways and lungs. The insertion tube is composed of a collection of minute glass fibers that have been coated with an optical insulation to provide light and image transmission. Since its development in 1966 and introduction to the United States in 1970, it has become a common application to a variety of diagnostic and therapeutic procedures that require direct visual examination or collection of pulmonary specimens.

Indications for Fiberoptic Bronchoscopy

✓ An example of diagnostic bronchoscopy is the evaluation of tumors in the airways and lungs.

Diagnostic bronchoscopy is done to gather additional information or to confirm preliminary diagnosis following history, physical, and other laboratory results (Holgate et al., 1992; Prakash et al., 1991; Prakash, 1994; Schuurmans et al., 2003). Biopsy and cytology samples are obtained for diagnostic purposes. Table 14-2 describes the techniques and application of flexible bronchoscopy. Indications for diagnostic bronchoscopy may include the evaluation of (1) tumors in the airways and lungs, (2) airway obstruction, (3) hemoptysis, inflammation and infection, (4) interstitial pulmonary disease, (5) staging of lung cancer before surgery, (6) vocal cord paralysis, and (7) tissue or fluid samples collected from the airways or lungs.

Therapeutic bronchoscopy may be used as a treatment modality because of the small size and versatility of the flexible bronchoscope. The general indications for therapeutic bronchoscopy include (1) removal of retained secretions, mucus plugs, or polyps in the

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TABLE 14-2 Application of Flexible Bronchoscopy

	Technique	Explanation
BIOPSY	Biopsy within visual range of the instrument	Biopsy is performed of targeted sites by forceps inserted through the bronchoscope.
	Transbronchial lung biopsy (TBLB)	Diseases appearing as diffuse shadows on chest X-rays include miliary tuberculosis, pneumoconiosis sarcoidosis, and various viral pneumonias, but it is almost impossible to obtain a definitive diagnosis based on X-ray findings only; therefore it is necessary to obtain tissue from the lung to establish a definitive diagnosis.
	Transbronchial needle aspiration biopsy (TBAB)	This method is employed when the lesion is located beyond the bronchial wall and there is no lesion in the bronchial lumen. The technique involves pressing the tip of the fiberscope lightly against the target puncture site, then firmly projecting the tip of the needle through the wall at that site. Then, while moving the tip backwards and forwards and from side to side, aspiration is performed. Then the needle is removed and the specimen is expelled on a slide by positive pressure from the syringe.
CYTOLOGY	Brushing for cytological specimens	If there is no protrusion in the bronchial lumen or if for some other reason the lesion is difficult to biopsy, a brush can be inserted through the instrument to obtain cytological material.
	Cytological brushing under fluoroscopic monitoring. This is used for the diagnosis of solitary lesions in the lung field.	First of all the related bronchi are defined by tomography or bronchography. Either forceps or a brush is inserted through the instrument channel and brought up to the target bronchus after inserting under direct endoscopic observation, thereafter bringing the forceps or brush up to the lesion under X-ray television monitoring.
	Bronchial Alveolar Lavage (BAL)	This involves wedging the tip of the flexible bronchoscope into the target segmental or subsegmental bronchus, flushing with physiologic saline solution, and then retrieving the liquid in order to examine it for cells washed from the surface of the bronchial mucosa or analysis of cellular components. Furthermore, washing of larger bronchi is referred to as bronchial lavage (BL).
	Bronchial Toilet	In cases that have undergone various thoracic surgical procedures, including cardiovascular or esophageal procedures, difficulty is often experienced in the expectoration of sputum. In such cases the flexible bronchoscope is employed routinely for the aspiration of bronchial secretions.
	Local administration of thrombin in cases of bloody sputum or hemoptysis	In cases of bloody sputum or hemoptysis, the bronchoscope is inserted, blood is aspirated, and if necessary, washing with physiologic saline is performed. Once the bleeding bronchus is verified the patient is placed lying on that side, to prevent aspiration of blood by the contralateral lung. Hemostasis can be achieved by insertion of a polyethylene tube and instillation of thrombin solution.

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TREATMENT

Removal of airway foreign bodies	While some believe that the rigid bronchoscope is useful in such cases, it is possible to remove almost all airway foreign bodies by means of employing a variety of grasping forceps. Foreign bodies removal in this way include gold crowns, peanuts (frequent in children), paper clips, drug containers, and fishbone.
Electrosurgical treatment	Joule heat caused by the electrosurgical current evaporates cells for incision and coagulation. Can be used for much of the same cases as Lasers, and treatment capability is expanded with the variety of accessories.

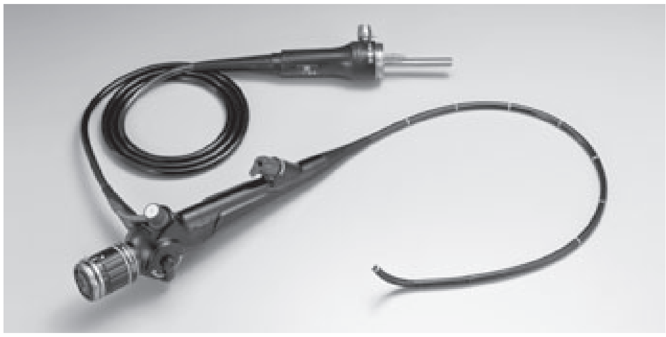
Courtesy of Olympus America Inc.

airway, (2) removal of foreign bodies (common in pediatric patients), (3) removal of endobronchial tissues, (4) intubation of difficult airway and (5) drainage of an abscess. Large foreign objects in the airway are removed by rigid bronchoscope.

✓ An example of therapeutic uses of fiberoptic bronchoscopy is the removal of secretions, mucus plugs, or polyps in the airway.

Bronchoscope and Medications

The main components of a fiberoptic bronchoscope are the handle and insertion tube (Figure 14-8). The handle consists of the eyepiece, bending mechanism, and



Specifications

Optical System	Field of view	120°
	Depth of Field	3–50 mm
Insertion Tube	Distal End Outer Diameter	ø 6.1 mm
	Insertion Tube Outer Diameter	ø 6.2 mm
	Working Length	550 mm
Instrument Channel	Channel Inner diameter	ø 3.2 mm
	Minimum Visible Distance	5 mm
Bending Section	Angulation Range	UP 180°, DOWN 130°
Total Length		840 mm

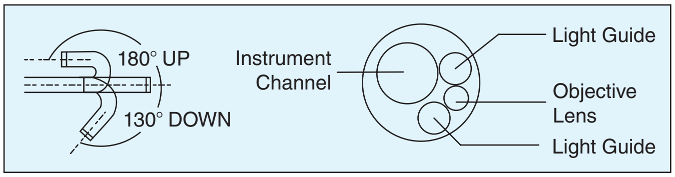


Image Courtesy of Olympus America Inc.

FIGURE 14-8 An example of flexible fiberoptic bronchoscope and specifications (Olympus model BF-XT40).

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TABLE 14-3 Medications for Bronchoscopy

Medication (Route)	Dosage (Route)	Purpose
Lidocaine	5 to 10 mL of 1 to 4% solution (aerosol)	Given 30 to 90 min before procedure to reduce irritation of the mucosal membrane caused by the insertion tube
Atropine sulfate	0.5 to 1.0 mg (IM)	Given before the procedure to reduce vagal response, oral secretions, and bronchospasm
Morphine sulfate	1.5 to 10 mg (IM)	Given before the procedure to provide pain relief and suppress coughing
Diazepam*	2.5 to 10 mg (IV bolus) or 10 to 15 mg (oral)	Given before and during the procedure as needed to provide sedation

*Sedation may also be induced by using (a) midazolam and fentanyl or (b) fospropofol disodium.

(Data from Bose et al., 2008; Gompertz et al., 1997; Jantz, 2009; Prakash et al., 1991; Williams et al., 1998.)

channel outlet. A video scope may be fitted onto the eyepiece for video taping of the procedure. The bending mechanism allows the physician to curve the insertion tube up (180°) or down (130°) for viewing of an anatomical structure at different angles. It also helps to direct the tube toward an intended segment or subsegment. The channel outlet is used for installation of saline or topical anesthetics, suction, and for passage of biopsy forceps, cytology brush, or cannula (Olympus America, Inc.).

Depending on the patient requirement and physician preference, four medications are commonly administered before the procedure. **Lidocaine** (5 to 10 mL of 1% to 4% solution) is given to the patient via aerosol 30 to 90 min before the procedure to reduce irritation of the mucosal membrane during the procedure. Before the procedure, **atropine sulfate** (0.5 to 1.0 mg) and morphine sulfate (1.5 to 10 mg) are administered intramuscularly. Atropine reduces vagal response, oral secretions, and bronchospasm. Morphine sulfate provides pain relief and suppresses coughing during the procedure. Diazepam or a suitable benzodiazepine is given intravenously in bolus to provide sedation. Table 14-3 summarizes the medications for bronchoscopy.

✓ Lidocaine, atropine sulfate, morphine sulfate, and diazepam are four common medications for bronchoscopy.

lidocaine: A medication used to reduce irritation of the mucosal membrane caused by the insertion tube.

atropine sulfate: A medication used to reduce vagal response, oral secretions, and bronchospasm during bronchoscopy.

Insertion of Bronchoscope

After testing the bronchoscope assembly, the distal end of the insertion tube is coated with a water-soluble lubricant and inserted via the nare, mouth (with bit block), endotracheal tube, or tracheostomy tube. Once the insertion tube enters the trachea and reaches above the carina, the tube is directed to the intended bronchi and segments (Figure 14-9).

For patients who are breathing spontaneously and without an artificial airway, oxygen therapy of up to 6 L/min may be given. If the bronchoscope insertion tube is inserted via an artificial airway (endotracheal or tracheostomy tube), an aerosol setup may be used. Adequate SpO_2 may be titrated with a pulse oximeter.

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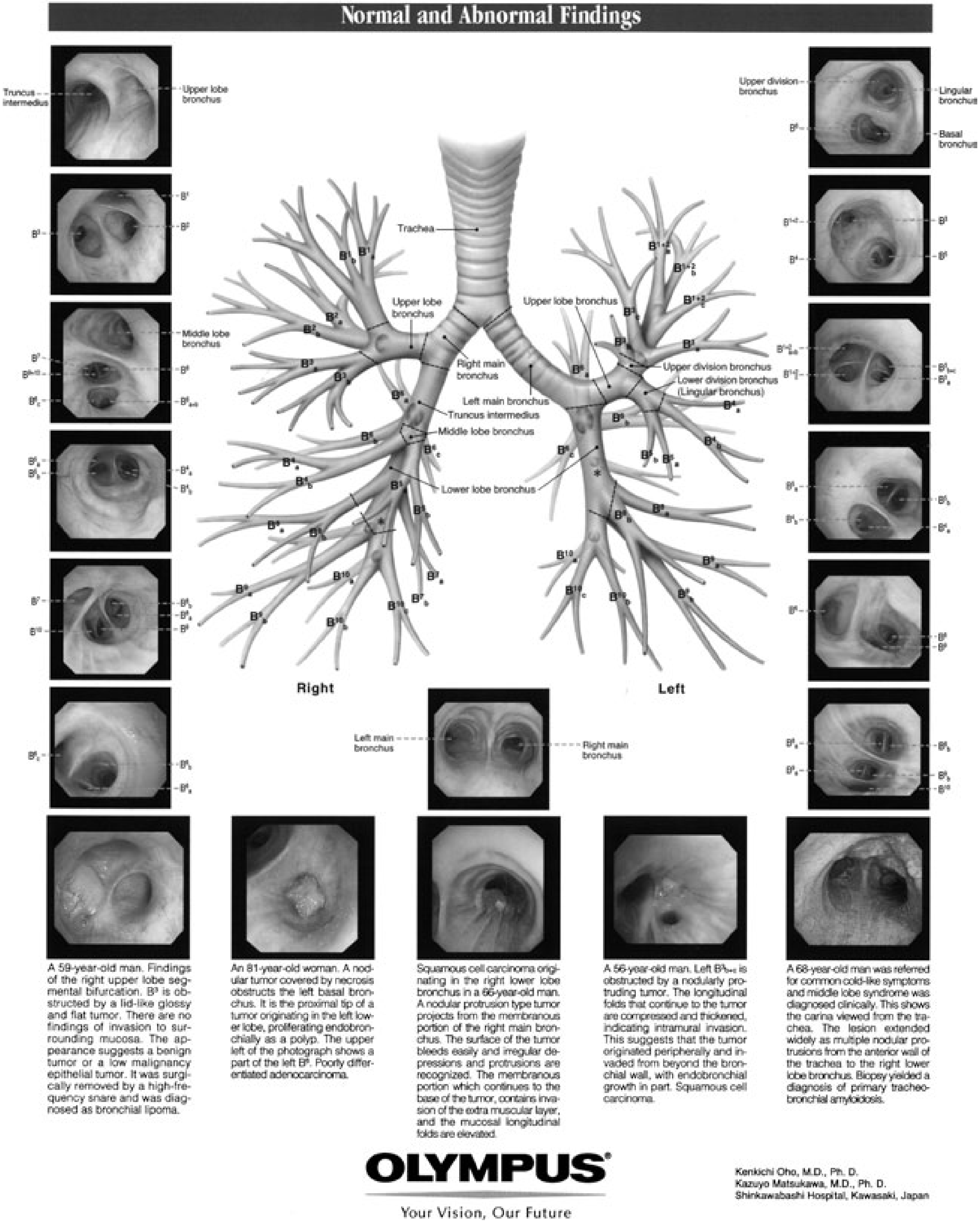
Normal and Abnormal Findings
Truncus intermedius
Upper lobe bronchus
Middle lobe bronchus
Upper division bronchus
Lingular bronchus
Basal bronchus
Trachea
Upper lobe bronchus
Right main bronchus
Left main bronchus
Truncus intermedius
Middle lobe bronchus
Lower lobe bronchus
Upper division bronchus
Lower division bronchus (Lingular bronchus)
Right
Left
Left main bronchus
Right main bronchus
OLYMPUS
Your Vision, Our Future

A 59-year-old man. Findings of the right upper lobe segmental bifurcation. B^3 is obstructed by a lid-like glossy and flat tumor. There are no findings of invasion to surrounding mucosa. The appearance suggests a benign tumor or a low malignancy epithelial tumor. It was surgically removed by a high-frequency snare and was diagnosed as bronchial lipoma.

An 81-year-old woman. A nodular tumor covered by necrosis obstructs the left basal bronchus. It is the proximal tip of a tumor originating in the left lower lobe, proliferating endobronchially as a polyp. The upper left of the photograph shows a part of the left B^6. Poorly differentiated adenocarcinoma.

Squamous cell carcinoma originating in the right lower lobe bronchus in a 66-year-old man. A nodular protrusion type tumor projects from the membranous portion of the right main bronchus. The surface of the tumor bleeds easily and irregular depressions and protrusions are recognized. The membranous portion which continues to the base of the tumor, contains invasion of the extra muscular layer, and the mucosal longitudinal folds are elevated.

A 56-year-old man. Left B^8_{b+c} is obstructed by a nodularly protruding tumor. The longitudinal folds that continue to the tumor are compressed and thickened, indicating intramural invasion. This suggests that the tumor originated peripherally and invaded from beyond the bronchial wall, with endobronchial growth in part. Squamous cell carcinoma.

A 68-year-old man was referred for common cold-like symptoms and middle lobe syndrome was diagnosed clinically. This shows the carina viewed from the trachea. The lesion extended widely as multiple nodular protrusions from the anterior wall of the trachea to the right lower lobe bronchus. Biopsy yielded a diagnosis of primary tracheobronchial amyloidosis.

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Image Courtesy of Olympus America Inc.

FIGURE 14-9 The normal bronchi and segments through the bronchovideoscope.

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✔ Hypoxia and increase in peak inspiratory pressure are common when bronchoscopy is done on patients receiving mechanical ventilation.

For patients receiving mechanical ventilation, an adaptor for the bronchoscope may be used. Minor air leak may be compensated by increasing the tidal volume. The pressure limit may need to be increased to make allowance for a larger tidal volume and insertion tube in the endotracheal tube.

Types of Specimen

In addition to pulmonary secretions, a flexible bronchoscope can collect other special specimens. Examples of these specimens are biopsy (forceps biopsy, transbronchial lung biopsy, and transbronchial needle aspiration biopsy) samples and bronchial brushing samples. Typically, biopsies collect tissue samples for examination, whereas bronchial brushing with or without alveolar lavage collects samples for cytological examination (Hattotuwa et al., 2002; Olympus America, Inc. 2003).

Secretion Collection. Pulmonary secretions are sometimes required for microscopic, culture and sensitivity, or cytology studies. These secretions are suctioned with a vacuum source and collected in a sterile sputum specimen trap. If the secretions are thick, pulmonary lavage with a saline solution may be necessary.

forceps biopsy: Tissue specimens collected by the forceps located at the distal end of the insertion tube.

Forceps Biopsy. **Forceps biopsy** is done within the visual range of the bronchoscope. Tissue specimens are obtained by passing the biopsy forceps through the biopsy channel outlet (Figure 14-10A). The razor-sharp biopsy device at the distal end of the forceps can be opened and closed by the operator using the control handle. The tissue specimen collected by the forceps is retrieved and put in a formalin solution for laboratory analysis.

transbronchial lung biopsy (TBLB): Tissue specimens collected by the forceps during a forced exhalation maneuver.

Transbronchial Lung Biopsy. **Transbronchial lung biopsy (TBLB)** is sometimes necessary to obtain a definitive diagnosis based on preliminary radiographic findings. Lung specimens are obtained by using a similar approach as in forceps biopsy. The patient is asked to inhale maximally, and the opened forceps are advanced deep into the distal airway near the target site. The patient then exhales maximally causing compression of the lung tissue around the biopsy forceps. The opened forceps are then closed by force. After removal of the forceps, the specimen is retrieved for laboratory analysis.

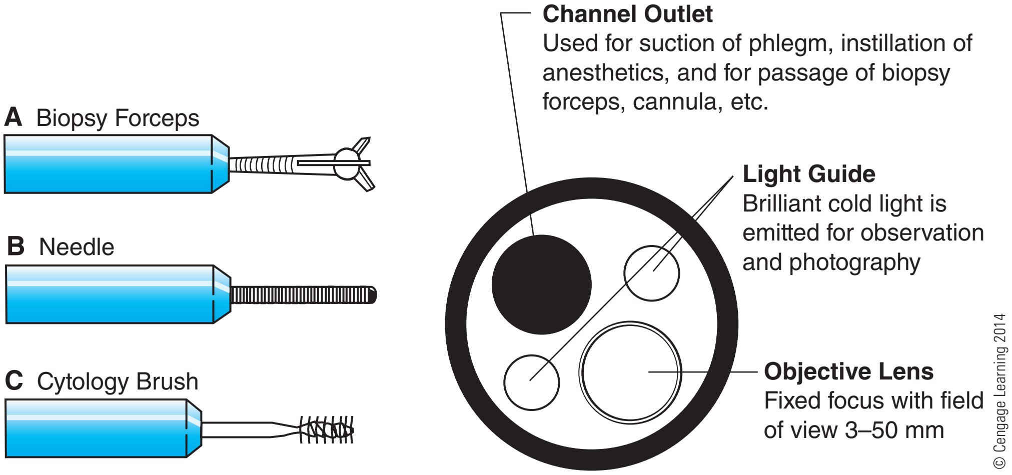
Channel Outlet
Used for suction of phlegm, instillation of anesthetics, and for passage of biopsy forceps, cannula, etc.
A Biopsy Forceps
Light Guide
Brilliant cold light is emitted for observation and photography
B Needle
Objective Lens
Fixed focus with field of view 3–50 mm
C Cytology Brush
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FIGURE 14-10 The distal end of the insertion tube; accessories for collection of samples using a-flexible bronchoscope: (A) Forceps; (B) Needle; and (C) Brush.

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transbronchial needle aspiration biopsy (TBNAB): Tissue specimens collected by applying aspiration while moving the needle at the sample collection site.

Transbronchial Needle Aspiration Biopsy. **Transbronchial needle aspiration biopsy (TBNAB)** is done where the lesion is located beyond the bronchial wall and there is no lesion in the bronchial lumen. This is done by pressing the tip of the bronchoscope gently against the target puncture site. The tip of the needle is then firmly projected through the mucosal wall. Aspiration (suction) is applied while moving the tip of the insertion tube of the bronchoscope back and forth and from side to side. The specimen (inside the needle) and the needle are retrieved from the bronchoscope and a syringe is used to expel the specimen from the needle (Figure 14-10B).

bronchial brushing: Tissue or loosened cell specimens collected by a shielded small brush using a brushing motion.

Bronchial Brushing. Cytologic examination may be necessary when an area of pathologic change is encountered during bronchoscopy. A shielded small brush is used to brush along the bronchial mucosa and the loosened cells are adhered to the brush (Figure 14-10C). The brush is then withdrawn into the shield and the entire apparatus is removed from the channel outlet of the bronchoscope. A microscopic slide may be made by fixing it with a suitable solution. An alternative is to cut off the brush and send it for pathologic tests.

Complications

✓ Complications of bronchoscopy include infection, hypoxemia, hemorrhage, and pneumothorax.

In vast majority of cases, bronchoscopy is a safe procedure. However, complications do occur and they range from infection to puncture of the lungs (Geraci et al., 2007; Pue et al., 1995).

Infection. There were reports of transmission and outbreak of *Mycobacterium tuberculosis* and *Pseudomonas aeruginosa* infections caused by the flexible bronchoscope (Agerton et al., 1997; Michele et al., 1997; Spach et al., 1993; Srinivasan et al., 2003). An increase in the frequency of *Pseudomonas* and other infections was also found to be associated with bronchoscopy (Hanson et al., 1991). These experiences show the need for diligent infection-control measures in the use of the bronchoscope.

Since the same bronchoscope is used to enter and explore different lung segments, the incidence of cross-contamination of healthy lung segments by infected ones is a possibility. Likewise, the saline solution used for lavaging the lung segments also poses a threat of cross-contamination of healthy lung segments.

Hypoxemia. The bronchoscope decreases the size of the airway opening and may cause partial airflow obstruction (Shakespeare et al., 2003). Ventilation/perfusion (V/Q) mismatch and secondary hypoxemia may occur during and after bronchoscopy because of the retained lavage solution, hypoventilation from premedication, mobilized and pooled secretions, and excessive suctioning. Supplemental oxygen should be used to alleviate the problem of secondary oxygen desaturation during bronchoscopy (Yildiz et al., 2002). In most cases, oxygen therapy may be discontinued 4 hours after bronchoscopy.

Hemorrhage. Bleeding can occur during bronchoscopy. Most minor bleeding may be stopped by saline lavage. When substantial bleeding occurs, a vasopressor (e.g., 1 mL of 1:1000 epinephrine with 9 mL of normal saline given in 2-mL portions) can be used to control bleeding from the biopsy site. Bleeding may also be stopped by wedging the bleeding site with the distal end of the bronchoscope. If wedging cannot be done because the airway is larger than the distal end of the bronchoscope, a 6-Fr Fogarty embolectomy catheter with balloon tip may be inserted through the channel. Wedging of the bleeding site may then be done by inflating the balloon tip of the catheter. Bronchoscopes that have a built-in coagulation electrode may stop hemorrhaging by using the coagulation mode (heat-induced coagulation).

Pneumothorax. Biopsy instruments are sharp and long enough to reach and puncture the periphery of the lung. In extremely rare cases, pneumothoraces may occur during bronchoscopy (Sun et al., 2003). Puncture of the lung while the patient is receiving mechanical ventilation may lead to tension pneumothorax. The vital signs of the patient must be monitored closely. Worsening cyanosis in spite of high oxygen concentration, diaphoresis, tachypnea, tachycardia, and thready pulse are some signs of tension pneumothorax. Chest tube is the treatment for tension pneumothorax and a complete chest tube setup should be on the bronchoscopy supplies list.

Postbronchoscopy Complications. Incidence of serious complications and mortality associated with fiberoptic bronchoscopy is low. In one survey of over 24,000 procedures, major complications occurred in 0.08% of all cases. The mortality rate was 0.1% to 0.2%, most of them secondary to hemorrhage and pneumothorax (Pue et al., 1995). If sudden deterioration of the patient's condition occurs, chest radiography should be taken to rule out pneumothorax.

✓ Hypoxemia and arrhythmias are the most common complications during and after bronchoscopy.

Hypoxemia and arrhythmias are the most common complications during and after bronchoscopy (Yildiz et al., 2002). Oxygen therapy, proper use of topical anesthesia, and use of intermittent suction are useful techniques to minimize the occurrence of these complications. Continuous pulse oximeter should be used during the recovery period.

Postbronchoscopy Care

Following bronchoscopy, the patient's vital signs are monitored for at least 2 hours or until stable. During the recovery period, an aerosolized bronchodilator may be used to treat bronchospasm. Oxygen is titrated to maintain a satisfactory SpO_2 and it may be provided via nasal cannula or continuous aerosol. Food and drink are withheld from the patient until the gag reflex has returned.

Cleaning and Care of Equipment. All equipment and supplies used in bronchoscopy must be cleaned, disinfected, or sterilized where indicated (Culver et al., 2003; Alvarado et al., 2000; Weber et al., 2001). The bronchoscope must also be maintained on a regular basis (AORN, 2001). The portion of the bronchoscope that comes in contact with the mucosal membrane must be sterilized. Bronchoscopes used in examining patients with tuberculosis or hepatitis B should also be sterilized. Ethylene oxide is used to sterilize delicate parts that cannot withstand steam autoclave. A checklist should be maintained to track delivery of equipment and supplies. Restocking of the disposables and consumables in the cart should be documented.

TRANSPORT OF MECHANICALLY VENTILATED PATIENTS

Patients receiving mechanical ventilation are critically ill or have deficiencies in the major organs or systems. They may also have difficulty in maintaining an adequate level of ventilation and oxygenation to fulfill their metabolic needs. In an intensive care setting, numerous resources are available to care for the mechanically ventilated patients. When the patients need to go from the intensive care setting to another location, some of these resources may not be readily available during transport. For this reason, a complete transport plan must be developed well in advance. The plan should include the transport team (e.g., ambulance or flight crew, RT, and nurse), procedure, and equipment and supplies necessary for a safe and uncomplicated transport.

Indications

The most common indication for intrahospital transport of mechanically ventilated patients is to acquire diagnostic procedures at a location other than the intensive care setting (e.g., MRI in the radiology department). For interhospital ground or air transport, it is usually done to acquire tertiary medical care or procedures (e.g., burn care, third-level nursery) that are only available at the destination facility (Ehrenwerth et al., 1986). For safety reasons, transport of mechanically ventilated patients must follow a thorough evaluation of the risk-benefit factor based on the patient's clinical condition.

Contraindications

✔ A patient should not be transported when the hemodynamic status is deteriorating or unstable.

Contraindications for transport of mechanically ventilated patients include inability to provide adequate oxygenation and ventilation, inability to maintain acceptable hemodynamic status, inability to provide adequate airway control or cardiopulmonary monitoring, and lack of trained transport team members (Weg et al., 1989). A patient should not be transported when the hemodynamic status is deteriorating or unstable.

Equipment and Supplies

The availability of medical equipment and supplies during transport is rather limited, and sometimes they may not be accessible. This is mainly due to limited space in the transport unit and partly due to unforeseen circumstances. This problem may be minimized by developing and using a list of equipment and supplies based on the type of transport and the characteristics of the patients. Table 14-4 shows a sample of essential respiratory care equipment and supplies for a typical transport.

Types of Transport

transport ventilator: A mechanical ventilator capable of operation without piped-in gas sources or electrical connection.

interhospital transport: Moving a patient between two hospitals.

Transport of patients who are being mechanically ventilated imposes many challenges, including: use of **transport ventilator** and emergency supplies, availability of a team of trained personnel, and distance of transport (Reynolds et al., 1992). When transporting a patient away from a medical care facility **(interhospital transport),** the transport team must decide which mode of transportation is most suitable. One main deciding factor is the distance between the departing facility and the destination facility. Four modes of transportation are available: ground ambulance, helicopter, propeller-drive aircraft, and jet (Rouse et al., 1992). There are advantages and disadvantages associated with each mode of transportation (Table 14-5).

Intrahospital Transport. Transport of patients within the hospital is done for the patient to receive advanced diagnostic procedures that require use of bulky or stationary equipment (e.g., CAT scan, MRI scan). For patients who require ventilatory

TABLE 14-4 Respiratory Care Equipment and Supplies for Transport of Mechanically Ventilated Patients

Type	Items*
Standard Precaution**	Gloves (sterile and clean) Gowns Eye protection
Patient Assessment and Monitoring	Stethoscopes Pulse oximeter Blood pressure cuffs Cardiac or hemodynamic monitor
Airway Management	Oropharyngeal and nasopharyngeal airways Resuscitation bags and masks Intubation supplies (laryngoscope handles and blades, fresh batteries, endotracheal tubes, etc.) Cuff manometer and 10-mL syringe Suctioning devices (catheters and tonsil tip)
Oxygenation Supplies	Oxygen cylinders (full and with wrench) Oxygen tubing with adaptor
Advanced Cardiac Life Support (ACLS) Pharmacological Agents	Drugs and solutions according to current ACLS Guidelines

**Some items require different sizes.*

***(Siegel et al., 2007).*

TABLE 14-5 Modes of Transportation

Mode	Range	Advantage	Disadvantage
Ground ambulance	<150 miles	Low cost, safer, and more reliable than helicopter	Limited by road and traffic conditions
Helicopter	<150 miles	Not limited by road and traffic conditions	Noisy, must have landing pad at both facilities
Propeller-driven aircraft	100 to 200 miles	Faster than helicopter if landing locations are near both facilities	Reduced P_IO_2 and relative humidity since low altitude aircrafts are not pressurized
Jet	>200 miles	Fast and able to travel intercontinental distance	Variable delivered V_T due to low or changing cabin pressure (commonly pressurized to 5,000 ft to 8,000 ft above sea level).

(Data from Cunningham et al., 1997; Mills et al., 2011; Nicholl et al., 1994; Shehey, 1995; Rouse et al., 1992.)

support for a short time (e.g., less than 30 min), manual ventilation with a resuscitation bag and oxygen may be sufficient. However, manual ventilation often causes inadvertent hyperventilation, irregular tidal volume and frequency, and fatigue of the care provider (Gervais et al., 1987; Hurst et al., 1989).

✓ Manual ventilation often causes inadvertent hyperventilation, varying tidal volume and frequency, and fatigue of the care provider.

intrahospital transport: Moving a patient between two locations within the hospital.

✓ For intrahospital transport of mechanically ventilated patients, a transport ventilator is preferable to manual ventilation.

For **intrahospital transport** of mechanically ventilated patients, a transport ventilator is preferable to manual ventilation (Nakamura et al., 2003). There are a number of simple and complex transport ventilators available on the market (e.g., Avian and LTV1000 by CareFusion, San Diego, CA; Crossvent 2 by Bio-Med Devices, Inc., Guilford, CT; Esprit by Respironics, Vista, CA; Model 740 by Covidien-Nellcor Boulder, CO; and Impact 754 Eagle Uni-Vent by Impact Instrumentation, Inc. West Caldwell, NJ). These FDA-approved transport ventilators are all battery-powered and have similar performance and functions of the current ventilators used in the ICU (Austin et al., 2002; Miyoshi et al., 2000).

✓ In pressure control mode, the expired volume must be monitored closely. Decreasing compliance or increasing airflow resistance can lead to decreasing tidal volume.

Besides the common features of a ventilator, the following capabilities are desirable on a transport ventilator: PEEP, pressure support ventilation, volume- and pressure-controlled ventilation, inverse ratio ventilation, display of expired volume, and visual and audible alarms, especially for disconnection and airway pressures. For ventilators that operate with only the pressure control mode, the expired volume must be monitored closely. Decreasing compliance or increasing airflow resistance can lead to decreasing tidal volume (Rola, 2004).

Ground Ambulance and Helicopter. Interhospital transport of mechanically ventilated patients can be done safely if the patient's medical condition is optimized before departure (Reynolds et al., 2002). A ground ambulance or helicopter may be used for patient transport when the distance is less than 150 miles. Each mode of transportation has its own advantages and disadvantages (Cunningham et al., 1997; Nichols et al., 1994; Shehey, 1995). Helicopters are preferable when the traffic or terrain condition precludes a timely arrival at the destination by ground ambulances. Of course, both facilities must have a suitable landing site for helicopters.

✓ The projected total time of transport should be a factor in deciding which mode of transportation to use.

Sometimes a propeller-driven aircraft is used for a distance between 100 and 200 miles. Transporting the patient from the departure facility to the takeoff location and from the landing location to the destination facility may be time-consuming. The projected total time of transport should be a factor in deciding which mode of transportation to use.

Jet. For distances greater than 200 miles or intercontinental transport, a jet is preferred. Use of a jet is typically done for nonemergency cases since a jet involves more resources and higher costs. The condition of patients using a jet for transport is rarely critical due to the time required for planning and long flight time. Typically, most patients use jets for transport to tertiary care facilities for advanced diagnostic procedure and therapeutic care, or from abroad to their home country (Veldman et al., 2001).

Procedures for Interhospital Transport

A written physician order must be obtained prior to transporting a mechanically ventilated patient. Before departure, equipment and supplies for the patient are

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gathered and assembled. For equipment using rechargeable batteries such as ventilator and monitor, maintain a full charge with electrical power until time for departure. For other equipment such as pulse oximeter and laryngoscope handle, new batteries should be installed. Monitors are calibrated and appropriate alarm limits set based on the patient's condition. Small "E" size oxygen cylinders with wrench must be full. In the event of delay, the oxygen content should be at least twice the volume needed for a one-way transport.

✓ During transport, the patient is monitored throughout the entire period to ensure stable hemodynamic status, adequate oxygenation, and ventilation.

All tubes and lines should be secured prior to transport. During transport, the patient is monitored throughout the entire period to ensure stable hemodynamic status, adequate oxygenation, and ventilation. All ventilator parameters are checked and adjusted to ensure proper functioning. A log should be kept to document all monitoring data, adverse reactions, and interventions.

On arrival, bedside monitors and ventilators at the destination hospital are used. The position and function of the patient's endotracheal tube are checked and documented. Pretransport ventilator settings and appropriate alarm limits are set on the ventilator after the patient has been stabilized from the transport. Finally, a verbal report is given to the therapist at the destination hospital, and the written documentation is filed with the patient's medical record.

Hazards and Complications

There are many hazards and complications associated with transport of mechanically ventilated patients. They range from hyperventilation during manual ventilation en route to a remote location to equipment failure. Table 14-6 summarizes the potential complications during transport and solutions for remediation.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) produces images for detection of abnormalities in the body organs not distinctively assessable by conventional imaging techniques. The strong magnetic field generated by the MRI scanner may draw certain metal objects toward the scanner. Steel oxygen transport cylinders and many ventilators are potential sources of a projectile risk to the patient and others. For this safety reason, only aluminum oxygen cylinders should be used (Morgan et al., 2002). All transport equipment coming near the MRI scanner must be resistant to the magnetic field produced by the scanner.

✓ Transport equipment coming near the MRI scanner must be resistant to the magnetic field produced by the scanner.

Some transport ventilators are capable of providing mechanical ventilation safely in an area where MRI is in use. Some MRI-compatible ventilators are IC-2A (MRI)® and MVP-10 (MRI)® (Bio-Med Devices, Guilford, CT); pNeuton Model A and Model S (Airon, Melbourne, FL); Omni-Vent® (Allied Healthcare Products, Inc., St. Louis, MO), MAXO$_2$Vent, (Maxtec, Salt Lake City, UT); and RespirTech PRO® (Vortran Medical Technologies, Sacramento, CA). When pressure control mode is used, the delivered volume may decrease in conditions of decreasing compliance or increasing airflow resistance. Monitoring the expired tidal volume is essential during transport with a mechanical ventilator.

TABLE 14-6 Potential Complications with Patient Transport and Solutions for Remediation

Hazard and Complication	Cause	Solution
Hyperventilation or variable tidal volumes	Manual ventilation with resuscitation bag	Use transport ventilator
Loss of PEEP/CPAP	Inaccuracy of PEEP valve on manual resuscitation bag	Use transport ventilator
Unstable hemodynamic status	Patient condition or excessive movement of the patient	Evaluate and stabilize hemodynamic status before transport
Loss of mechanical ventilation or monitoring capability	Equipment failure	Provide manual ventilation with oxygen
Inadvertent extubation or removal of vascular access lines	Movement of patient without paying attention to arrangement of equipment/supplies	Coordinate movement of the patient and equipment/supplies
Power/gas source failure	Various causes	Use ventilator with battery capability and provide portable compressed oxygen source
Hypoxia due to high-altitude hypobaric condition	High altitude in nonpressurized aircraft	Monitor ventilation and SpO_2; use a higher F_IO_2; use pressurized aircraft

(Data from Beckmann et al., 2004; Farmer, 1996; Dockery et al., 1999; McGough et al., 1992; Waydhas et al., 1995.)

Transport ventilators are typically smaller and have fewer features than traditional ventilators. In one report, a full-function (Servo 300) ventilator has been modified and used safely near an MRI scanner (Morgan et al., 2002).

SUMMARY

This chapter provides an overview of the essential elements of three procedures that are done to mechanically ventilated patients: chest tube and drainage system, fiberoptic bronchoscopy, and transport of mechanically ventilated patients. In a critical care setting, these procedures often involved a team of health care providers, and respiratory therapists play a major role on the team. For this reason, respiratory therapists must be proficient with these procedures. A good working knowledge of these procedures is also essential for efficient teamwork and patient safety.

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Self-Assessment Questions

1. Indications for chest tube placement include all of the following *except*:

A. tension pneumothorax.	C. chest trauma.
B. pleural effusion.	D. hemothorax.

2. The sharp pointed instrument for incision into the chest cavity for chest tube placement is called a:

A. trocar.	C. needle.
B. chest tube.	D. blade.

3. The ideal location of chest tube placement for drainage of pleural fluid is at the:

   A. second to third intercostal space at the midaxillary line.
   B. fourth to sixth intercostal space at the midaxillary line.
   C. second to third intercostal space at the midclavicular line.
   D. fourth to sixth intercostal space at the midclavicular line.

4. During chest tube insertion, Dr. Farland enters the chest wall by going ________ the rib because ________.

   A. above, blood vessels and nerves lie below the rib
   B. above, this location is easier to puncture
   C. below, blood vessels and nerves lie above the rib
   D. below, this location is easier to puncture

5. In a three-chamber drainage system, the amount of suction applied to the pleural space is determined by the ________ in the suction control chamber.

   A. water level and submersion depth of the venting tube
   B. amount of pleural fluid in the chamber
   C. wall vacuum level
   D. all of the above

6. A flexible bronchoscope may perform all of the following *except*:

   A. visual inspection of the airway.
   B. collection of specimens.
   C. suction.
   D. removal of large tumor.

7. Dr. Kingston asks a therapist to prepare the patient for bronchoscopy and to reduce the irritation to the mucosal membrane caused by the tube. The therapist should administer ________ via aerosol nebulizer about ________ before the procedure.

A. atropine sulfate, 10 min	C. lidocaine, 10 min
B. atropine sulfate, 30 min	D. lidocaine, 30 min

8. Atropine sulfate is used to ________ during bronchoscopy.

   A. provide pain relief
   B. reduce vagal response, oral secretions, and bronchospasm
   C. minimize irritation to the mucosal membrane
   D. provide sedation

9. During bronchoscopy, ________ may be used to collect tissue biopsy samples.

   A. saline lavage
   B. suction catheter
   C. special needle and forceps
   D. brush

10. In order to recognize the common complications promptly during and after bronchoscopy, the patient's ________ should be monitored closely.

    A. respiratory frequency and SpO_2
    B. blood pressure and respiratory frequency
    C. cardiac rhythm and pulmonary artery pressure
    D. SpO_2 and cardiac rhythm

11. A patient is being ready for transport to a special-care hospital 100 miles away. The transport team may choose from all of the following modes of transportation *except*:

    A. ground ambulance.
    C. jet.
    B. helicopter.
    D. propeller-driven aircraft.

12. During patient transport, hyperventilation and variable tidal volumes are two common undesirable outcomes when a(n) ________ is used.

    A. anesthesia bag
    B. manual ventilation device
    C. volume control mode of ventilation
    D. pressure control mode of ventilation

Answers to Self-Assessment Questions

1. C.
2. A.
3. B.
4. A.
5. A.
6. D.
7. D.
8. B.
9. C.
10. D
11. C.
12. B.

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Additional Resources

Chest Tube

Carroll, P. (1996). Salvaging blood from the chest. *RN Journal, 59*(9), 3–8.

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Chapter 15

Critical Care Issues in Mechanical Ventilation

David W. Chang

Outline

Introduction
Acute Lung Injury and Acute Respiratory Distress Syndrome
  Definitions of ALI and ARDS
  Pathophysiology
  Clinical Presentations
  Lung Protection Using Airway Pressure Thresholds
  Low Tidal Volume and Permissive Hypercapnia
  Decremental Recruitment Maneuver to Determine Optimal PEEP
  Prone Positioning
Ventilator-Associated Pneumonia
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Hypoxic-Ischemic Encephalopathy
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  Cerebral Perfusion Pressure
  Decrease in CPP Due to Cardiac Arrest
  Decrease in CPP Due to Shock
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  Delayed Brain Injury
  Acceleration and Deceleration Brain Injuries
  Clinical Evaluation and Assessment
  Management Strategies
  Respiratory Management
Summary
Self-Assessment Questions
Answers to Self-Assessment Questions
References

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Key Terms

acute lung injury (ALI)
acute respiratory distress syndrome (ARDS)
cerebral perfusion pressure (CPP)
clinical pulmonary infection score (CPIS)
decremental recruitment maneuver
Glasgow coma scale (GCS)
hypoxic-ischemic encephalopathy (HIE)
lung protection strategy
permissive hypercapnia
prone positioning
subglottic secretion drainage
transtentorial herniation
ventilator-associated pneumonia (VAP)

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Use the clinical criteria to differentiate between ALI and ARDS.
- Describe the management of ALI and ARDS using airway pressure thresholds, low tidal volume, permissive hypercapnia, recruitment maneuver, and prone positioning.
- Outline the clinical signs, prevention, and treatment of ventilator-associated pneumonia.
- List the factors that lead to hypoxic-ischemic encephalopathy.
- Describe the management of hypoxic-ischemic encephalopathy.
- Outline the clinical signs and respiratory management of traumatic brain injury.

INTRODUCTION

Mechanical ventilation is frequently used to correct and support hypoventilation and hypoxemia in a variety of clinical conditions. While a mechanical ventilator is a frequently used device, its application is highly dependent on the patient's clinical and physiologic conditions. This chapter provides an overview of some critical care issues that are closely related to mechanical ventilation.

ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME

acute lung injury (ALI): A condition of sudden onset, characterized by non-cardiogenic pulmonary edema on chest radiograph and a PaO_2/F_IO_2 of ≤ 300 mm Hg.

One complication of prolonged mechanical ventilation is induced lung injury due to overdistention and repetitive recruitment and derecruitment (opening and closing) of noncompliant lung units (Dreyfuss et al., 1998). In nonhomogenous lung diseases such as **acute lung injury (ALI)** and **acute respiratory distress syndrome (ARDS),**

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acute respiratory distress syndrome (ARDS): A condition of sudden onset, characterized by non-cardiogenic pulmonary edema on chest radiograph and a PaO_2/F_IO_2 of ≤ 200 mm Hg.

✓ When non-homogenous lungs are ventilated by positive pressure, the non-compliant units are recruited intermittently while the compliant units suffer from over-distention.

normal compliant lung units are mingled with collapsed noncompliant lung units. When these lungs are ventilated by positive pressure, the noncompliant units are recruited intermittently while the compliant units suffer from overdistention. As early as 1970, a study showed that when a peak transalveolar pressure of 30 cm H_2O was applied to the nonhomogenous lung units, the stress on the wall between normal and collapsed units reached 140 cm H_2O (Mead, 1970). This pressure harms the compliant lung unit since they cannot sustain extreme high pressures.

Definitions of ALI and ARDS

Acute respiratory distress syndrome (ARDS) is a more severe form of acute lung injury (ALI). The mortality rate of patients with ARDS ranges from 10% to 90%. This wide range may be attributed to the nonuniformity of the disease processes, definition for ARDS, therapy modality, and patient population. In 1992, the American-European Consensus Conference on ARDS recommended four evaluation parameters for the classification of ALI and ARDS. These parameters are timing of onset, oxygenation, chest radiograph, and pulmonary capillary wedge pressure (PCWP) (Bernard et al., 1994). Table 15-1 lists the criteria and thresholds for the classification of ALI and ARDS.

TABLE 15-1 Criteria and Thresholds to Classify ALI and ARDS

Evaluation Parameter	ALI	ARDS	Notes
1. Timing	Acute onset	Acute onset	
2. Oxygenation	$PaO_2/F_IO_2 \leq 300$ mm Hg (regardless of PEEP level)	$PaO_2/F_IO_2 \leq 200$ mm Hg (regardless of PEEP level)	*Effects of PEEP are time-dependent*
3. Chest Radiograph	Bilateral infiltrates on frontal chest radiograph		*Review pulmonary capillary wedge pressure (PCWP) to evaluate cause of pulmonary edema*
4. Pulmonary Capillary Wedge Pressure (PCWP)	≤18 mm Hg when measured or no clinical evidence of left atrial hypertension		*To rule out cardiogenic pulmonary edema*

(Bernard et al., 1994.)
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ARDS is a more severe form of ALI. The PaO_2/F_IO_2 threshold for ALI is ≤ 300 mm Hg, and for ARDS it is ≤ 200 mm Hg.

The criteria for ALI and ARDS are same with the exception of the PaO_2/F_IO_2 values ($\leq$300 mm Hg for ALI and $\leq$200 mm Hg for ARDS—regardless of PEEP level for ALI and ARDS). When using the PaO_2/F_IO_2 as an evaluation tool for ALI and ARDS, it is essential to consider other conditions that may contribute to the decrease of PaO_2/F_IO_2. For example, hypoventilation can lower the PaO_2 *and* PaO_2/F_IO_2.

Pathophysiology

Direct injury to the lungs can lead to pathological abnormality in the intra-alveolar space and alveolar filling by edema, fibrin, collagen, neutrophilic aggregates, or blood.

The alveolar-capillary membrane consists of the alveolar epithelium and the vascular endothelium. Research studies have shown that damage to the lungs can occur on either side of the membrane.

Direct Injury. Exposure of alveolar epithelium to direct insult (e.g., bacteria) leads to activation of alveolar macrophages and development of inflammatory response within the lungs. Furthermore, alveolar epithelial damage can lead to pathological abnormality in the intra-alveolar space and alveolar filling by edema, fibrin, collagen, neutrophilic aggregates, or blood (Johanson et al., 1985; Paolo et al., 1993; Rocco et al., 2008). The radiographic signs of ALI typically show infiltrates and pulmonary consolidation. Clinical conditions that are associated with *direct* lung injury and development of ARDS are shown in Table 15-2.

Indirect injury to the lungs primarily lead to microvascular congestion and interstitial edema, with relative sparing of the intra-alveolar spaces.

Pneumonia, aspiration, and inhalation of toxins are some conditions that can cause *direct* lung injury.

Sepsis, severe trauma, and acute pancreatitis are some conditions that can cause *indirect* lung injury.

Indirect Injury. An indirect insult to the lungs originates from the action of inflammatory mediators released from extrapulmonary foci into the systemic circulation. Once in the systemic circulation, the first target of damage is the pulmonary vascular endothelial cell, with an increase of vascular permeability and recruitment of monocytes, polymorphonuclear leukocytes, platelets, and other cells. This type of pathological alteration due to an indirect insult is primarily microvascular congestion and interstitial edema, with relative sparing of the intra-alveolar spaces (Muller-Leisse et al., 1993; Paolo et al., 1993; Rocco et al., 2008). Clinical conditions that are associated with *indirect* lung injury and development of ARDS are shown in Table 15-2.

TABLE 15-2 Clinical Conditions Associated with Development of ARDS

Direct Lung Injury	Indirect Lung Injury
Pneumonia	Sepsis
Aspiration	Severe trauma
Inhalation of toxins	Acute pancreatitis
Near drowning	Cardiopulmonary bypass
Pulmonary contusion	Transfusion of blood products
Fat embolism	Drug overdose

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Treatment modalities for many critical conditions tend to target corrections of the underlying causes (e.g., antibiotics for infection). At the present time, the best known and most common management strategy for ALI and ARDS is supportive care for oxygenation and ventilation. Studies on lung injury have identified risk factors and have suggested that certain critical care interventions may influence the incidence of lung injury. In the future, a well-designed screening tool or a lung injury predictive model may help to reduce the incidence of ALI and ARDS (Litell et al., 2011).

Clinical Presentations

✔ A combination of severe hypoxia, increased deadspace, decreased lung compliance, and patient fatigue contributes to the development of ventilatory failure.

✔ During the exudative phase of ALI and ARDS, chest radiographs reveal a progression from diffuse interstitial infiltrates to diffuse, fluffy, alveolar opacities.

✔ Patients with ARDS often lack cardiogenic signs of pulmonary edema such as cardiomegaly, pleural effusions, and vascular redistribution. The PCWP measurement should be normal when pulmonary edema is casued by ARDS.

In the early stage of ARDS, the clinical signs may include tachypnea, tachycardia, and mild hypoxemia. The patient's oxygenation status worsens due to V/Q mismatch and intrapulmonary shunting. The PaO_2/F_IO_2 ratio continues to decrease as ARDS progresses. Severe hypoxia becomes evident with increasing deadspace ventilation and decreasing lung compliance. When the patient cannot keep up with the increasing work of breathing and oxygen demand, the $PaCO_2$ begins to increase and progresses to severe respiratory acidosis. Most patients develop diffuse alveolar infiltrates and eventual respiratory failure within 48 hours of the onset of symptoms (Mortelliti et al., 2002).

Lung Imaging. During the exudative phase of ALI and ARDS, chest radiographs reveal a progression from diffuse interstitial infiltrates to diffuse, fluffy, alveolar opacities. Figure 15-1 shows the typical chest radiograph of a patient with ARDS. The appearance of infiltrates and opacities is typically bilateral. Reticular (crisscrossing lines) opacities on the chest radiograph suggest the development of interstitial fibrosis.

Although the radiographic signs of pulmonary edema caused by ARDS and congestive heart failure are similar, patients with ARDS often lack cardiogenic signs of pulmonary edema such as cardiomegaly, pleural effusions, and vascular redistribution. In addition, the PCWP measurement should be normal when pulmonary edema is caused by ARDS.

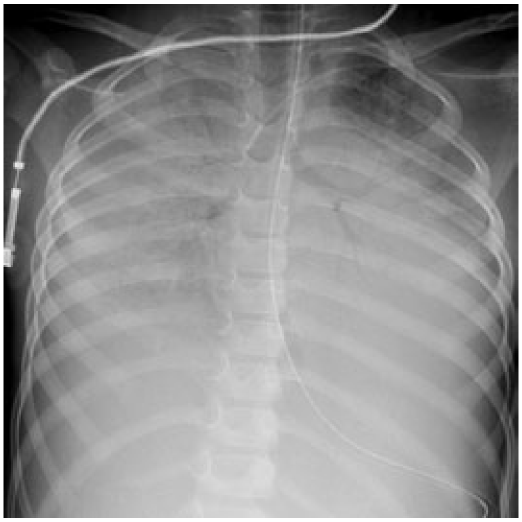

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FIGURE 15-1 Chest radiograph of ARDS.

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Lung Protection Using Airway Pressure Thresholds

Studies have shown that lung injuries during mechanical ventilation are associated with high airway pressures. Barotrauma or volutrauma is one of the severe complications of positive pressure ventilation. In patients with ARDS and reduced compliance, high peak inspiratory pressure is usually required because of the low lung compliance. The increase in airway pressures has the potential to injure the lung units that have normal or high compliance. Positive pressure ventilation can also cause lung injuries such as pneumomediastinum, pneumoperitoneum, pneumothorax, tension pneumothorax, and subcutaneous emphysema (Bezzant et al., 1994; Slutsky, 1994).

lung protection strategy: A method to prevent the lungs from pressure- or volume-induced injuries during mechanical ventilation.

Lung protection strategy is a method to prevent the lungs from pressure- or volume-induced injuries during mechanical ventilation. The general agreement of lung protection is to use the lowest pressures (i.e., PIP, P_{PLAT}, mPaw) or tidal volume possible. Studies recommend that, in most cases, the airway pressures should be kept as follows: peak inspiratory pressures <50 cm H_2O, plateau pressures <35 cm H_2O, mean airway pressures <30 cm H_2O, and PEEP <10 cm H_2O (Bezzant et al., 1994; Slutsky, 1994). (Note: In 2000, the ARDSNet recommended plateau pressure <30 cm H_2O.)

✓ Risk of barotrauma may be reduced by keeping the PIP <50 cm H_2O, P_{PLAT} <35 cm H_2O, mPaw <30 cm H_2O, and PEEP <10 cm H_2O. In 2000, the ARDSNet recommended plateau pressure <30 cm H_2O.

The suggested thresholds on airway pressures should be used as a *guideline*. Depending on the patient and other coexisting conditions, the pressure thresholds must be adjusted as indicated.

Low Tidal Volume and Permissive Hypercapnia

High airway pressure and high tidal volume increase the risk for ARDS in patients receiving mechanical ventilation for greater than 48 hours (Jia et al., 2008). Low tidal volume and **permissive hypercapnia** are two strategies that can partially minimize these risk factors. The primary advantage of using low tidal volume or permissive hypercapnia is to minimize the airway pressures and to reduce the risk for barotrauma (Feihl et al., 1994; Hall et al., 1987).

permissive hypercapnia: A lung protection method that uses low tidal volume (i.e., 4 to 7 mL/Kg) and allows the $PaCO_2$ to rise above 50 mm Hg.

Low Tidal Volume. In patients with high lung compliance, increased functional residual capacity and air trapping, barotrauma is likely due to preferential distribution of mechanical tidal volume to lung units with high compliance. Patients with COPD are candidates of acquiring overdistention, air trapping, and auto-PEEP during positive pressure ventilation. For these patients, the tidal volume should be reduced. The peak inspiratory flow should be increased to allow a longer expiratory time for adequate exhalation.

✓ For patients with COPD, the tidal volume should be reduced. The peak inspiratory flow should be increased to allow a longer expiratory time for adequate exhalation.

Mechanical ventilation with low tidal volume increases deadspace ventilation and decreases alveolar ventilation. Complications of the low tidal volume method include acute hypercapnia and respiratory acidosis, increased work of breathing, dyspnea, and atelectasis (Kallet et al., 2001a and 2001b).

✓ Mechanical ventilation with low tidal volume increases deadspace ventilation and decreases alveolar ventilation.

Permissive Hypercapnia. Permissive hypercapnia uses low tidal volume during volume-controlled ventilation and allows the $PaCO_2$ to rise above the upper-normal limit

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In permissive hypercapnia, the tidal volume is titrated until the PIP is near the plateau pressure measured before the low tidal volume procedure.

(e.g., $>$50 mm Hg). The tidal volume used in permissive hypercapnia is in the range of 4–7 mL/kg (Feihl et al., 1994). Since the plateau pressure (i.e., end-inspiratory occlusion pressure) is the best estimate of the average peak alveolar pressure, the tidal volume is titrated until the PIP is near the plateau pressure measured before the low tidal volume procedure. Using the plateau pressure as the target PIP avoids alveolar overdistention and reduces the likelihood of lung injury (Hall, 1987; Slutsky, 1994).

Since permissive hypercapnia provides minimal ventilation with lower tidal volume and pressure, it can be a safe mechanism to protect the lungs of patients with ARDS (Feihl et al., 1994; Hickling et al., 1990). (Refer to Chapter 12 for the mechanism and physiologic changes of permissive hypercapnia.)

Tromethamine (THAM) is a nonbicarbonate buffer that helps to compensate for metabolic acidosis by directly decreasing the hydrogen ion concentration and indirectly decreasing the carbon dioxide level. It is preferable to bicarbonate in patients undergoing permissive hypercapnia.

The elevated $PaCO_2$ and acidosis during permissive hypercapnia can lead to CNS dysfunction, increase in intracranial pressure, neuromuscular weakness, cardiovascular impairment, and increased pulmonary vascular resistance. These complications of permissive hypercapnia may be alleviated by returning the pH to its normal range, either by renal compensation over time or by neutralizing the acid with bicarbonate or tromethamine (a nonbicarbonate buffer) (Marini, 1993).

Decremental Recruitment Maneuver to Determine Optimal PEEP

ARDS and PEEP. For patients with ARDS, the potential for lung injury is high because different lung units have different pressure requirements. Lung units with low compliance require high opening and sustaining pressures. These high airway pressures can overstretch and injure the normal compliant lung units.

In mechanical ventilation, PEEP is used to prevent repetitive recruiting and derecruiting of the atelectatic portion of the lung (i.e., lung units with low compliance). PEEP also enhances alveolar ventilation, improves hypoxemia due to intrapulmonary shunting, and reduces total lung water. When PEEP is used in patients with ARDS, it carries many detrimental effects. The primary complication of PEEP is pressure-induced lung injury (due to increased mean airway pressure) and volume-induced lung injury (due to overdistension of alveoli). Since PEEP raises the peak inspiratory pressure and the combined pressure is transmitted to the pleural space, it increases the pulmonary vascular resistance, decreases left-ventricular compliance, venous return, cardiac output, and systemic oxygen delivery. The reduction in cardiac output in turn causes renal insufficiency, decreased urine output, and increased sodium and water retention (Kallet et al., 2007; kznhealth.gov, 2011). Due to these potential complications, the selection of an optimal PEEP is crucial.

The ARDSNet recommends keeping the $P_{PLAT} < 30$ cm H_2O with low tidal volume and using a combination of F_IO_2 and PEEP (See Table 15-3) to maintain O_2 sat $>$88%.

In 2000, the Acute Respiratory Distress Syndrome Network (ARDSNet) published the following initial ventilator settings and combination of F_IO_2 and PEEP for patients with ARDS (Table 15-3). Following initial setup and stabilization, subsequent PEEP levels may be titrated to obtain the optimal PEEP based on the patient's requirement.

Titration of Optimal PEEP. The methods to obtain an optimal PEEP have been undergoing changes over the years. Earlier methods used compliance, PaO_2, SpO_2, lung

TABLE 15-3 ARDSNET Initial Settings for Patients with ARDS

1. Volume-controlled ventilation
2. Assist/control mode
3. Keep $P_{PLAT} < 30$ cm H_2O (reduce V_T as low as 4 mL/Kg predicted body weight to reach this P_{PLAT} target)
4. Maintain SaO_2 or SpO_2 88% to 95%
5. Set the PEEP using the F_IO_2 /PEEP combination below to obtain O_2 sat > 88%

F_IO_2 (%)	30	40	50	60	70	80	90	100
PEEP (cm H_2O)	5	5–8	8–10	10	10–14	14	16–18	20–24

function (pulmonary oxygen transfer efficiency), and FRC (open-lung ventilation) as titration parameters and endpoints. Recently, ventilation homogeneity (total ventilation distribution) using electrical impedance tomography has been described as a titration method for the optimal PEEP (Kallet et al., 2007; Lachmann, 1992; Zhao et al., 2010).

Recent studies used a lung recruitment maneuver before PEEP titration. Incremental and decremental lung recruitment methods have been reported to determine the optimal PEEP for patients with ARDS. Table 15-4 outlines the **decremental recruitment maneuver** (Girgis et al., 2006).

decremental recruitment maneuver: A method of titration for optimal PEEP by setting a high CPAP (and PEEP) and gradually decreasing the pressure and F_IO_2.

Contraindications for Recruitment Maneuvers. Based on Meade et al. (2008), the patients who benefit most from recruitment maneuvers are those having the worst pulmonary edema and are most at risk of dying from refractory hypoxemia due to ALI or ARDS. Since only 10 to 15% of patients with ALI/ARDS die of refractory hypoxemia as the primary cause (most deaths are due to nonpulmonary organ failure), the routine use of recruitment maneuvers in unselected patients with ALI is not recommended (Stapleton, 2008).

Recruitment maneuvers produce extreme high peak airway pressure, plateau pressure, and PEEP. They should not be done to patients with existing barotraumas, compromised hemodynamic status, or presence of blebs or bullae on chest radiography. Increased intracranial pressure should be considered a contraindication for recruitment maneuvers (Kacmarek et al., 2007).

✓ Recruitment maneuver should be used on patients with severe pulmonary edema and who are most at risk of dying from refractory hypoxemia due to ALI or ARDS.

✓ Recruitment maneuvers should not be done to patients with existing barotraumas, compromised hemodynamic status, presence of blebs or bullae on chest radiography, and increased intracranial pressure.

Prone Positioning

Prone positioning is done by placing the bed and patient in a Trendelenberg position at 15 to 30 degrees. The prone position places the majority of the lower lobes in an uppermost position. This position reduces the opening pressure of the lower lobes, enhances the distribution of ventilation, and reduces the gradient of transpulmonary pressure across the lungs. This physiologic effect is beneficial for patients with severe gas exchanging impairment. Patients who require a PEEP >10 cm H_2O and $F_IO_2 \geq 60\%$ to maintain supine oxygen saturation of ≥90% are candidates of prone positioning (Marini et al., 2004). (See Chapter 12 for a discussion on prone positioning.)

prone positioning: A procedure to temporarily improve a patient's oxygenation by placing the bed and patient in a Trendelenberg position at 15 to 30 degrees.

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TABLE 15-4 Decremental Recruitment Maneuver (RM) to Determine Optimal PEEP

Step	Notes
1. Sedation	Until no spontaneous breathing effort (During procedure all vital signs are monitored and supported)
2. Continue sedation	Apply F_IO_2 100% for 20 min Do not disconnect patient from ventilator, use inline suction system
3. *Initial* Recruitment Maneuver (RM)	Apply F_IO_2 100% & CPAP 40 cm H_2O, up to 40 sec (Discontinue recruitment if one of the following occurs: $SpO_2 < 88\%$; HR > 140/min or < 60/min; MAP < 60 mm Hg or $> 20\%$ from baseline; arrhythmia)
4. Immediately after *initial* RM	Initiate PCV at 15 cm H_2O and PEEP at 20 cm H_2O (for a total of 35 cm H_2O of peak pressure) Go to next step if PaO_2 increases by 20% of the pre-RM value (RM may be repeated up to 3 times if PaO_2 does not increase by 20% of the pre-RM value)
5. Following *initial* RM	F_IO_2 is decreased gradually by 5% to 20% until SpO_2 stabilizes between 90% and 94%
6. Following *initial* RM	PEEP is decreased by 2 cm H_2O every 15 to 20 min until SpO_2 drops below 90%
7. Optimum PEEP	The PEEP immediately preceding the SpO_2 drops below 90%
8. *Final* RM	Apply F_IO_2 100% & CPAP 40 cm H_2O for 40 sec
9. After *final* RM	Initiate mechanical ventilation using F_IO_2 (from step 5) and optimal PEEP (from step 7)

(Girgis et al., 2006.)

VENTILATOR-ASSOCIATED PNEUMONIA (VAP)

ventilator-associated pneumonia (VAP): A severe form of hospital-acquired infection of the lung parenchyma that develops within 48 hours after intubation and initiation of mechanical ventilation.

Ventilator-associated pneumonia (VAP) is a severe form of hospital-acquired infection. It is defined as a newly acquired infection of the lung parenchyma that develops within 48 hours after intubation and initiation of mechanical ventilation.

Incidence of VAP

VAP affects 10 to 25% of mechanically ventilated patients. Approximately 25% of all infections acquired in intensive care units are due to VAP. The mortality rates of

✓ Risk factors for VAP include long duration of mechanical ventilation, advanced age, depressed level of consciousness, preexisting lung disease, immune suppression due to disease or medications, and malnutrition.

VAP range from 33 to 50%. The risk of VAP is highest immediately after intubation and initiation of mechanical ventilation. For the first 5 days, the incidence of VAP is 3%. The rate decreases to 2% per day for the next 5 days, and 1% per day thereafter. Patients who are admitted to the trauma, neurosurgical, or burn units have a higher incidence of VAP than those in the respiratory units and medical ICUs (Byrd et al., 2010; Cook et al., 1998; Craven, 2006).

Risk factors for VAP include long duration of mechanical ventilation, advanced age, depressed level of consciousness, preexisting lung disease, immune suppression due to disease or medications, and malnutrition (Torpy et al., 2008).

Clinical Presentations

✓ VAP is often associated with fever, leukocytosis or leukopenia, and purulent tracheobronchial secretions.

VAP is often associated with fever, leukocytosis or leukopenia, and purulent tracheobronchial secretions. The radiographic signs of VAP include new or progressive infiltrates on chest radiography, The presence of lung infiltrates plus two of the three criteria listed above had a sensitivity of 69% and a specificity of 75% for the diagnosis of VAP (Torres et al., 2004).

Depending on the onset of VAP, predominant microorganisms include flora of the upper airway, gram-negative bacilli and methicillin-resistant *S. aureus*. Table 15-5 outlines the common microbes during the course of VAP (Torres et al., 2004).

clinical pulmonary infection score: An objective scoring system to use as an additional aid in the diagnosis of ventilator-associated pneumonia (VAP) and decision on antimicrobial therapy.

A score of >6 in the modified **clinical pulmonary infection score** (CPIS) (Table 15-6) has been used as an additional aid in the diagnosis of VAP and in decisions on antimicrobial therapy. One study (Fartoukh et al., 2003) emphasized the difficulties of the clinical diagnosis of pneumonia in mechanically ventilated patients suspected of VAP. It suggested that the modified CPIS should be used cautiously in clinical practice, and further refinements of the clinical scoring approach (e.g., use of other biological markers of infection) are needed to improve the usefulness of the modified CPIS in the management of VAP. Laboratory samples obtained from bronchoalveolar lavage (BAL) fluid and protected specimen brush (PSB) are two examples of useful biological markers of infection (Mayhall, 2001).

✓ A modified CPIS score of more than 6 at baseline or after incorporating the gram stains or culture result is suggestive of pneumonia.

TABLE 15-5 Common Microbes during the Course of VAP

Onset of VAP After Intubation and Initiation of Mechanical Ventilation	Common Microbes
First 48 hours	Upper airway flora (e.g., *Haemophilus influenza* and *Streptococcus pneumonia*)
3 to 7 days	Gram-negative bacilli (e.g., *Pseudomonas aeruginosa, Escherichia coli, Acinetobacter, Proteus and Klebsiella* species)
>7 days	*Staphylococcus aureus* to include methicillin-resistant strain (MRSA)

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TABLE 15-6 The Modified Clinical Pulmonary Infection Score (CPIS)

CPIS Points*	0	1	2
1. Tracheal secretions	Rare	Abundant	Abundant + purulent
2. Chest X-ray infiltrates	No infiltrate	Diffused	Localized
3. Temperature, °C	36.5–38.4	38.5–38.9	<36 or >39
4. Leukocytes count, $1000/mm^3$	4–11	<4 or >11	<4 or >11 + >500 bands
5. PaO_2/F_IO_2, mm Hg	>240 or ARDS	<240 and no evidence of ARDS	
6. Microbiology	Negative		Positive

**Obtain Protected Alveolar Lavage (PAL) or Bronchial Alveolar Lavage (BAL) sample for Gram stain and culture prior to empiric antibiotic therapy (suctioned sputum is suboptimal but acceptable in select patients).*

The modified CPIS at baseline is calculated from the first five variables (1–5). A score of more than 6 at baseline or after incorporating the gram stains or culture results is suggestive of pneumonia. (Note: If the CPIS is <6, consider alternate diagnosis. CPIS has not been validated in immunosuppressed patients.)

Prevention of VAP

Prevention of VAP does not require an elaborate plan or extraordinary efforts. One hospital bundled several simple steps of prevention and reduced the incidence of VAP by 95% in three years. These steps include using proper cuff pressure to minimize the incidence of aspiration, changing the ventilator circuits every seven days, changing the inline suction devices and heat and moisture exchange filters every 24 hours, using special endotracheal tubes to suction pooled secretions in the subglottic region, and using single-use vials of saline for endotracheal lavage when indicated (Darves, 2005). (Note: saline lavage is a controversial practice.)

In addition to the prevention steps described above, other studies have been done to evaluate the best strategies for the prevention of VAP. They include elevation of the head of the bed at a 30- to 45-degree angle at all times (Smulders et al., 2002), changing ventilator circuits with HMEs only when malfunctioned or visibly soiled (Tablan et al., 2003), early weaning from mechanical ventilation, hand hygiene, aspiration precaution and prevention of contamination (Apisarnthanarak et al., 2007), decontamination of the oropharynx, noninvasive ventilation with face mask (Antonelli et al., 1998; Brochard et al., 1995; Hilbert et al., 2001), sedative vacation (temporary hold), use of oral feeding tube, use of oral chlorhexidine (Darves, 2005), endotracheal tube with an ultrathin and tapered-shape cuff membrane and coated in silver or antimicrobial agents, and endotracheal tubes with a separate dorsal lumen for **subglottic secretion drainage** (Kollef et al., 2008; Lorente et al., 2010; Valles et al.,1995). Table 15-7 summarizes the methods and the respective rationales in the prevention of VAP in mechanically ventilated patients.

✓ Methods to prevent VAP include: exercise good hand hygiene, elevate head of bed at 30- to 45-degree angle, change ventilator circuit when visibly soiled, use noninvasive ventilation, and schedule sedation vacation for 6 to 8 hours daily.

subglottic secretion drainage: This procedure uses a special endotracheal tube with a separate dorsal lumen for suctioning of secretions above the ET tube cuff.

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TABLE 15-7 Methods to Prevent VAP in Mechanically Ventilated Patients

Method	Rationale
1. Exercise good hand hygiene (wash hands with soap and water or use alcohol gel before and after patient contact; avoid wearing rings during work)	Reduce nosocomial infection
2. Elevate head of bed at 30- to 45-degree angle at all times	Reduce incidence of aspiration
3. Change ventilator circuit when visibly soiled or malfunctioned	Reduce incidence of contamination with water condensate or secretions during circuit change
4. Use noninvasive ventilation with face mask	Avoid direct access of pathogens to lower respiratory tract
5. Schedule sedation vacation for 6 to 8 hours daily	Allow weaning assessment and early extubation
6. Initiate early weaning from mechanical ventilation	Reduce exposure of lower airway to pathogens and ventilator-induced lung injuries
7. Follow sterile techniques in suctioning and prevent contamination of endotracheal or tracheostomy tube	Reduce exposure of lower respiratory tract to pathogens
8. Perform good oral care or decontaminate oropharynx	Reduce exposure of lower respiratory tract to pathogens
9. Use oral feeding tube	Use of nasal feeding tube may cause sinusitis, a condition associated with VAP
10. Use endotracheal tube with an ultrathin and tapered-shape cuff membrane	Reduce incidence of aspiration
11. Use endotracheal tube coated with silver or antimicrobial agents	Provide protection against some pathogens
12. Use endotracheal tube with a separate dorsal lumen above the cuff for subglottic secretion drainage	Reduce incidence of aspiration (*routine use* is not supported by reference Koenig et al., 2006)

Among the methods to prevent VAP, proper and frequent hand washing is by far the simplest and most cost-effective way to reduce the incidence of VAP. While research studies strongly support the elevation of head of bed, there are disagreements on the degree of elevation. Darves (2005) emphasized that the angle of elevation is not a critical issue and elevation should be done on mechanically ventilated patients unless the practice is contraindicated (e.g., postneurosurgical, severe hypotension).

Other methods have been described in the prevention of VAP. However, the effectiveness of these methods does not have universal agreement, and their use is controversial. More clinical studies may be needed to validate their use. In a 2006 publication, Koenig et al. do not support the *routine use* of ET tubes with subglottic suction capabilities, rotational beds, inline suction systems, rotational antibiotic schemes, or selective gut decontamination. Selective gut decontamination is a practice widely used in Europe in the prevention of VAP, but its effectiveness is not universal in the United States. The reason may be attributed to the regional drug resistance in the ICU (Darves, 2005). In the future, clinical experience and evidence may add to the knowledge of VAP and the methods for prevention of VAP.

Treatment of VAP

✓ In the management of VAP, most research studies recommend empiric broad-spectrum antibiotics that cover pathogens resistant to multiple drugs.

In the management of VAP, most research studies recommend empiric broad-spectrum antibiotics that cover pathogens resistant to multiple drugs. Modifications of drug therapy can be made after determination of the sensitivities of the causative organism. Knowledge of organisms that cause VAP (e.g., early- or late-onset VAP) in the individual ICU and the pattern of antibiotic resistance can help to choose the most appropriate and effective broad-spectrum antibiotics (Koenig et al., 2006; Torres et al., 2004).

✓ For patients already on antibiotics at the time of suspected VAP, the antibiotics chosen should be from different classes, as it is likely that resistance to "in-use" antibiotics has already developed.

✓ Any change in CPIS can guide clinical decisions on the continuing use, selection, or stoppage of antibiotics.

In general, for patients already on antibiotics at the time of suspected VAP, the antibiotics chosen should be from different classes, as it is likely that resistance to "in-use" antibiotics has already developed. Assessment of the presence of VAP should be repeated by day 3, using the modified clinical pulmonary infection score (CPIS) (Table 15-6). Any change in CPIS can guide clinical decisions on the continuing use, selection, or stoppage of antibiotics. Assessment of quantitative culture results and sensitivities is also important. This assessment provides selection and use of pathogen-specific antibiotics as well as the correct dosage. Improper use of antibiotics may lead to drug resistance and prolonged hospitalization. (Koenig et al., 2006).

Since the knowledge of VAP and the use of antibiotics for the treatment of VAP are evolving, readers should refer to the most recent research findings for the management of VAP.

HYPOXIC-ISCHEMIC ENCEPHALOPATHY (HIE)

hypoxic-ischemic encephalopathy (HIE): A condition caused by a severe lack of oxygen supply to the brain leading to damage to the cells and neurons of the brain and spinal cord.

cerebral perfusion pressure (CPP): The pressure required to provide blood flow, oxygen, and metabolite to the brain; it is a function of mean arterial pressure (MAP) and intracranial pressure (ICP); CPP = MAP − ICP.

Hypoxic-ischemic encephalopathy (HIE) is a condition caused by a severe lack of oxygen supply to the brain, leading to damage to the cells and neurons of the brain and spinal cord. Although the term often refers to injury sustained by newborns (injury or complication during birth), HIE can be used to describe any injury to the brain due to severe hypoxia (Kohnle, 2011). Three broad categories of acute cerebral hypoxia are: inadequate ventilation or oxygenation (e.g., respiratory arrest, carbon monoxide poisoning, drowning), inadequate perfusion (e.g., cardiac arrest, shock, blocked or ruptured blood vessels), and decrease in **cerebral perfusion pressure (CPP)** (e.g., decrease in mean arterial pressure or increase in intracranial pressure).

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General Principles of HIE

✔ Causes of acute cerebral hypoxia include inadequate ventilation or oxygenation, inadequate perfusion, and decrease in cerebral perfusion pressure.

The brain occupies about 2% of the total body weight but uses 15% of the energy generated by the body. Unlike the muscle cells in the body, the brain does not hold or store any energy of its own except for a small amount of glycogen in the astrocytes. Furthermore, the brain cells cannot utilize fatty acids because they are not transported across the brain capillaries. The brain is highly dependent on the constant supply of oxygen and glucose provided by the blood. For these reasons, a severe deprivation of oxygen (e.g., respiratory/cardiac arrest, shock, severe anemia, decrease in CPP) or lack of glucose (e.g., hypoglycemia, starvation) can severely affect the normal functions of the brain (Agamanolis, 2011).

✔ A lack of cerebral circulation depletes the neuronal oxygen stores within 20 seconds and leads to unconsciousness. Within 5 minutes of complete cerebral anoxia, brain glucose and ATP stores are lost, and energy depletion is the end result.

A lack of cerebral circulation depletes the neuronal oxygen stores within 20 seconds and leads to unconsciousness. Within 5 minutes of complete cerebral anoxia, brain glucose and ATP stores are lost, and energy depletion is the end result (Madl et al., 2004). The mechanism of cerebral cellular injury and death as a result of energy deprivation is complex. The first event following energy depletion is failure of the Na$^+$ and K$^+$ pumps, leading to depolarization of the neuronal membrane. Depolarization causes neurons to release excessive glutamate into the synaptic cleft. Since some glutamate receptors are nonselective cation-permeable ion channels, influx of excessive Ca^{++} into neurons leads to activation of catabolic (destructive) enzymes, as well as activation of nitric oxide synthase and increase of nitric oxide production. Furthermore, *passive* influx of Cl$^-$ (and Na$^+$) into the cells can cause osmotic edema and rapid death of the cells (Agamanolis, 2011). Figure 15-2 outlines the mechanism of cerebral cellular injury as a result of energy depletion to the brain.

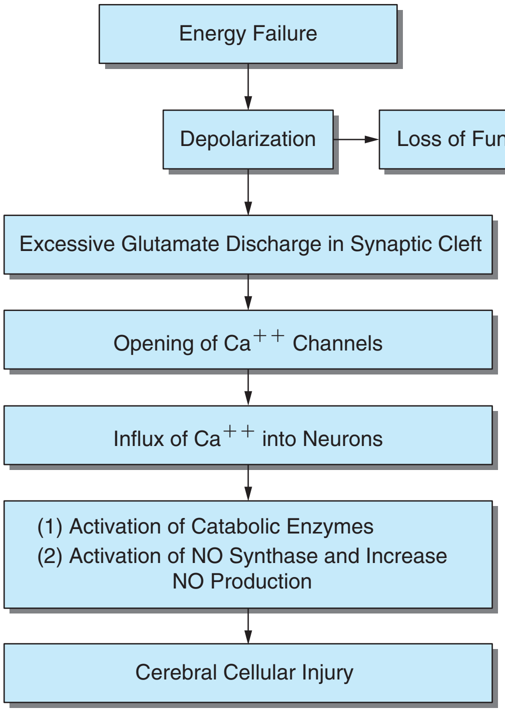
Energy Failure
Depolarization
Loss of Function
Excessive Glutamate Discharge in Synaptic Cleft
Opening of Ca^{++} Channels
Influx of Ca^{++} into Neurons
(1) Activation of Catabolic Enzymes
(2) Activation of NO Synthase and Increase NO Production
Cerebral Cellular Injury

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FIGURE 15-2 Mechanism of cerebral cellular injury as a result of energy depletion to the brain.

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✓ CPP = MAP − ICP

The most common cause of energy depletion and failure is a drop in cerebral perfusion (global ischemia). Cerebral perfusion is governed by the cerebral perfusion pressure (CPP). In turn, the CPP is the difference between the mean arterial pressure (MAP) and intracranial pressure (ICP). CPP = MAP − ICP. Perfusion-related conditions (e.g., cardiac arrest, hypotension) are conditions that cause a decrease in MAP and a direct reduction in CPP and cerebral perfusion. Non-perfusion-related conditions (e.g., traumatic brain injury, increase in ICP) cause an indirect reduction in CPP and cerebral perfusion.

Symptoms. In mild cases of HIE, symptoms range from difficulty concentrating or paying attention, poor judgment or coordination, euphoria, and extreme lethargy. In more advanced or severe cases of cerebral oxygen deprivation, seizures and coma may result (Kohnle, 2011). In respiratory care, most patients with lung diseases and hypoxia may exhibit signs of mild HIE. They usually respond to oxygen therapy and other respiratory therapy very well. The following sections discuss the topics related to severe cerebral hypoxia.

Cerebral Perfusion Pressure

✓ Inadequate cerebral perfusion can cause a wide range of neurological problems ranging from cerebral ischemia to brain death.

Cerebral perfusion pressure (CPP) is the pressure required to provide blood flow, oxygen, and metabolites to the brain. Under normal conditions, the brain regulates its own blood flow regardless of the systemic blood pressure and cerebral vascular resistance. However, the brain becomes vulnerable in conditions of severe hypotension (e.g., cardiac arrest). The autoregulation ability of the brain may also be lost following head trauma, whereas the cerebral vascular resistance is often greatly elevated. Depending on the severity of reduction in cerebral perfusion, neurological effects on the brain may range from cerebral ischemia to brain death (Bouma & Muizelaar, 1990; Marion et al., 1991).

✓ The normal cerebral perfusion pressure (CPP) should range between 70 and 80 mm Hg. Mortality rate increases about 20% for each 10 mm Hg drop in CPP.

There is no class I evidence for the optimum level of CPP, but the critical threshold is believed to be from 70 to 80 mm Hg. The mortality rate increases about 20% for each 10 mm Hg drop in CPP. In studies involving severe head injuries, a 35% reduction in mortality was achieved when the CPP was maintained above 70 mm Hg (Bouma et al., 1992; Rosner et al., 1990).

Decrease in CPP Due to Cardiac Arrest

Cardiac arrest and shock are two common causes of severe hypotension. Based on a 2002 World Health Organization report, the incidence of cardiac arrest is estimated between 36 and 128 per 100,000 persons per year. CPR was performed in 86% of these cases with a successful return of spontaneous circulation in 17 to 49% of these resuscitated individuals. However, about 80% of patients who initially survive a cardiac arrest remain in a coma for different durations, about 40% remain in a persistent vegetative state, and 80% are dead at one year. (Madl et al., 2004).

Hypotension as a result of cardiac arrest is essentially a mechanical failure, wherein the pump (heart) is not generating adequate pressure to provide perfusion.

Advanced cardiac life support is the primary method to manage cardiac arrest. Therapeutic hypothermia (32 to 34°C) has been used to lower the oxygen utilization and to improve the neurologic outcomes in cardiac arrest victims (Madl et al., 2004). Treatments of severe hypotension should be aimed at the underlying causes. For cardiac arrest, restoration of the heart function is the primary goal. Use of ACLS, oxygen, antiarrhythmics, beta agonists, fluid, and vasopressors are essential procedures to manage severe hypotension due to cardiac arrest.

Decrease in CPP Due to Shock

✔ The CPP may become inadequate when the MAP is too low.

✔ Severe hypotension can impair neurological functions. A drop in MAP will lower the CPP (↓CPP = ↓MAP − ICP).

✔ ICP control is typically not necessary because ICP tends to stay below its clinical threshold (i.e., < 20 mm Hg).

✔ The CPP can be maintained above the critical threshold by raising the MAP.

Hypotension as a result of shock is usually due to lack of circulating volume. This deficiency may be due to severe blood loss (absolute hypovolemia) or vasodilatation (relative hypovolemia, as in septic shock). As in cardiac arrest, hypotension causes abnormally low systolic, diastolic, and mean arterial pressures (MAP). Since CPP is the difference between the MAP and ICP, a drop in MAP will lower the CPP (↓CPP = ↓MAP − ICP).

Based on the relationship of MAP and ICP, a higher CPP may be maintained by raising the MAP or by lowering the ICP. In clinical practice, ICP control is typically not necessary because ICP tends to stay below its clinical threshold (i.e., <20 mm Hg) under normal conditions. In conditions of severe hypotension, CPP may become suboptimal. The CPP can be maintained above the critical threshold by *raising* the MAP (Changaris et al., 1987; Rosner & Daughton, 1990).

In the absence of hemorrhage, the MAP should be managed initially by maintaining an adequate fluid balance. It may then be followed by using a vasopressor such as norepinephrine or dopamine. Systemic hypotension (SBP <90 mm Hg) should be avoided and controlled as soon as possible because early hypotension is associated with increased morbidity and mortality following severe brain injury (Chesnut et al., 1993; Marmarou et al., 1991).

Decrease in CPP Due to Brain Injury

✔ An increase in ICP (e.g., traumatic brain injury) can reduce the CPP and blood supply to the brain (↓CPP = MAP − ↑ICP).

Traumatic brain injury raises the ICP due to swelling of the brain within a confined fixed space (skull). The increase in ICP reduces the CPP and blood supply to the brain (↓CPP = MAP − ↑ICP). The end result is energy depletion and development of HIE.

Evaluation and Treatment of HIE

The severity of anatomic and physiologic changes in the brain and spinal cord may be evaluated by examining the structure and function of the brain. The tests may include CT or MRI scan, EEG, ultrasound, and evoked potential test (analysis of brain wave). These tests may also be used to evaluate the effectiveness of treatments for HIE (Kohnle, 2011).

Treatments for HIE depend on the underlying cause, as well as the severity of the damage to the brain. Treatment options typically focus on ventilation, perfusion, and seizure control. Hypothermia using cooling blankets has been used to reduce oxygen consumption and to minimize the effects of cerebral hypoxia. Hyperbaric oxygen therapy increases the oxygen-carrying capacities of the plasma and is most useful in conditions with increased dysfunctional hemoglobins such as carbon monoxide poisoning.

TRAUMA BRAIN INJURY

The major causes of traumatic brain injury (TBI) include motor vehicle crashes, falls, assaults, sports-related injuries, penetrating trauma, and explosive blasts and combat injuries. The male-to-female ratio for TBI is about 2-to-1, and most patients with TBI are younger than 35 years old. While motor vehicle crashes cause most deaths and injuries to young people, falls are the leading cause of death and disability from TBI in people older than 65 years (Ghajar, 2000).

✓ The normal ICP is 8 to 12 mm Hg (the clinical normal is up to 20 mm Hg).

Almost 100% of patients with severe head injury and two-thirds of those with moderate head injury become permanently disabled in performing some daily functions. In the U.S., there are about 600,000 new TBI cases per year. The direct cost of caring for these patients after discharge from the hospital is estimated to be $25 billion annually (Crippen, 2011).

✓ ICP $>$ 20 mm Hg reduces CPP and increases the likelihood of cerebral hypoxia or ischemia and death.

✓ The clinical normal range for CCP is 70–80 mm Hg. A suboptimal CPP ($<$70 mm Hg) can cause cerebral hypoxia or ischemia and death.

The brain is contained within the skull, a rigid and nonelastic structure which has a capacity of about 1,500 mL. Under normal conditions, the brain occupies 85 to 90% of this intracranial compartment. The intravascular cerebral blood volume and cerebrospinal fluid account for about 10% and 3% of this volume, respectively. The brain has a very low compliance and cannot tolerate significant rapid volume expansion (e.g., cerebral edema, hematoma). Significant volume expansion within a rigid skull will cause the intracranial pressure (ICP) to rise. The normal range for ICP is 8–12 mm Hg (the clinical normal is up to 20 mm Hg). An increase of ICP higher than 20 mm Hg will lower the CPP ($\downarrow$CPP = MAP $-$ $\uparrow$ICP). The clinical normal range for CCP is 70–80 mm Hg. A suboptimal CPP ($<$70 mm Hg) can cause cerebral hypoxia or ischemia and death.

Delayed Brain Injury

Neurological damage does not always occur at the moment of impact (primary injury). The events that happen afterwards (secondary injury) may lead to brain swelling, increase in intracranial pressure (ICP), and decrease in CPP and cerebral blood flow (Irwin et al., 2003). Ischemia of the brain tissues is the end result when CPP cannot provide adequate perfusion to the brain. Secondary neurological injury to the brain is the leading cause of in-hospital deaths following primary TBI (Marshall et al., 1991). Another risk factor for secondary injury is systemic hypotension. Systemic hypotension lowers the MAP and CPP ($\downarrow$CPP = $\downarrow$MAP $-$ ICP).

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Acceleration and Deceleration Brain Injuries

A direct blow to the head causes *acceleration* brain injury. This type of injury causes the skull to move *away* from the blow. Since the brain does not move away from the blow at the same speed as the skull, the skull makes impact to the brain and causes brain injury. In *deceleration* brain injury, the skull makes impact to a stationary object (e.g., a fall on concrete). The skull stops suddenly when it makes impact on the concrete. The brain continues to move toward the concrete and make impact with the skull. In both acceleration and deceleration injuries, the various cellular components of the brain or the axons are injured. In some cases, rotational forces applied to the skull and brain can also cause shear injuries on the axons (Crippen, 2011).

Explosive blasts are a common cause of TBI in active military personnel. Researchers believe the pressure wave from explosives passing through the brain directly disrupts brain function (Mayoclinic, 2012). In addition to the pressure wave, TBI may also result from acceleration (e.g., shrapnel) and deceleration (e.g., falls or head collisions with stationary objects) brain injuries.

Brain Herniation. Within the skull, the two dural structures (falx cerebri and tentorium cerebella) divide the intracranial compartment into three sections. These two dural structures have central openings with prominent edges at the borders of these openings. When the ICP becomes significantly elevated, the brain can become herniated and slide through these openings within the falx or tentorium. Brain injury often occurs when the brain makes contact with the dural edge (Crippen, 2011). There are five types of brain herniation and each type has its distinct characteristics. Transtentorial herniation is one example and it is discussed below. Readers should refer to a neurology reference source for other types of brain herniation.

transtentorial herniation: A type of brain injury that causes the downward displacement of the medial aspect of the temporal lobe (uncus) through the tentorial notch by a mass above.

Transtentorial herniation is a type of brain injury that causes the downward displacement of the medial aspect of the temporal lobe (uncus) through the tentorial notch by a mass above. This condition exerts pressure on the underlying structures, including the brainstem and the third cranial nerve. Compression of the third cranial nerve suppresses the parasympathetic input to the eye, resulting in a dilated pupil. A unilateral (one side only) dilated pupil is a classic sign of transtentorial herniation of the brain. Eighty percent of the dilated pupil occurs ipsilateral (same side) to the side of herniation. If a unilateral dilated pupil is not observed, unilateral posturing can be a sign of transtentorial herniation. Unilateral dilated pupil or/and unilateral posturing requires immediate evaluation for surgical intervention (trauma.org[1], 2011).

Clinical Evaluation and Assessment

Glasgow coma scale (GCS): A scoring system to determine the degree of traumatic brain injury; score of 13 to 14 (mild), score of 9 to 12 (moderate), score of 3 to 8 (severe).

TBI is graded on the basis of the level of consciousness (Table 15-8) or **Glasgow coma scale (GCS)** score (Table 15-9). The level of consciousness ranges from cloudy consciousness (mild) to brain death (most severe). Mild TBI has a GCS score of 13 to 14. Moderate injury has a score of 9 to 12, and severe injury has a score of 3 to 8 (Teasdale et al., 1974).

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TABLE 15-8 Assessment of Impairment Due to Traumatic Brain Injury

Term	Finding
Cloudy consciousness	A mild deficit in processing information by the brain Short-term memory is diminished but long-term memory remains intact
Lethargy	Decrease in alertness, rouse briefly in response to stimuli, impaired ability to perform task, aware of surrounding, return to inactivity when left alone
Obtundation	Decrease in awareness and alertness, rouse briefly in response to stimuli, unable to perform task, unaware of surrounding, return to inactivity when left alone
Stupor	Cannot communicate clearly but can be aroused by painful stimulation
Coma	Does not respond to vigorous stimulations
Brain death	Irreversible cessation of whole brain function as documented by EEG

(Crippen, 2011.)

Management Strategies

In mild TBI, rest and pain relievers are usually sufficient to treat a headache. The patient is typically monitored closely at home and scheduled with a follow-up medical appointment for any persistent or worsening symptoms. In moderate to severe TBI, oxygenation and circulatory supports are essential. Diuretics, antiseizure drugs, and coma-inducing drugs may be used as indicated to reduce intracranial pressure and preserve brain function (mayoclinic.com, 2012).

The primary goal in the management of severe TBI is to prevent secondary neuronal injury and to avoid further loss of neurons. This condition should be managed by the following procedures (trauma.org[1], 2011):

1. Protect airway
2. Provide eucapnic ventilation and adequate oxygenation to correct cerebral hypoxia
3. Correct hypovolemia and hypotension to provide adequate systolic and mean arterial pressures
4. CT scan as needed to evaluate severity and response to treatments
5. Neurosurgery as indicated to treat swelling and edema, and to reduce elevated ICP
6. Intensive care monitoring and management

Systolic pressure should be kept above 90 mm Hg (Brain Trauma Foundation, 2011). Hypovolemia and hypotension may be managed by control of hemorrhage and fluid administration. A CT scan of the head can be helpful to determine the presence of epidural or subdural hematoma. A surgical decision can be made based on the size of intracranial lesion.

TABLE 15-9 Glasgow Coma Scale

		Eye Opening	
Score	**1 year to adult**	**<1 year**	
4	Spontaneous	Spontaneous	
3	To verbal command	To shout	
2	To pain	To pain	
1	No response	No response	
		Best Motor Response	
Score	**1 year to adult**	**<1 year**	
6	Obeys command		
5	Localizes pain	Localizes pain	
4	Flexion withdrawal	Flexion withdrawal	
3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)	decorticate: arms flexed or bent inward on the chest, clenched fists, legs extended and feet turned inward.
2	Extension (decerebrate)	Extension (decerebrate)	decerebrate: head arched back, arms extended by the sides, legs extended
1	No response	No response	
		Best Verbal Response*	
Score	**5 years to adult**	**2–5 years**	**<2 years**
5	Oriented and converses	Appropriate words	Cries appropriately
4	Disoriented and converses	Inappropriate words	Cries
3	Inappropriate words; cries	Screams	Inappropriate cry/scream
2	Incomprehensible sounds	Grunts	Grunts
1	No response	No response	No response

**Verbal scores cannot be assessed in intubated patients. The maximal score for intubated patients is 10T and the minimal score is 2T (T is used to designate intubation).*

Modified from: Crippen, D.W. (2011).

Respiratory Management

✓ To manage TBI during mechanical ventilation, the $PaCO_2$ may be titrated to a level as low as 26 torr during the first 24 hours of mechanical ventilation.

Patients with severe TBI should be sedated to prevent coughing or ventilator-patient dyssynchrony because these conditions tend to increase the intracranial pressure. Endotracheal suctioning should be avoided or the frequency of suctioning should be minimized. If mechanical ventilation is used, the $PaCO_2$ may be reduced to

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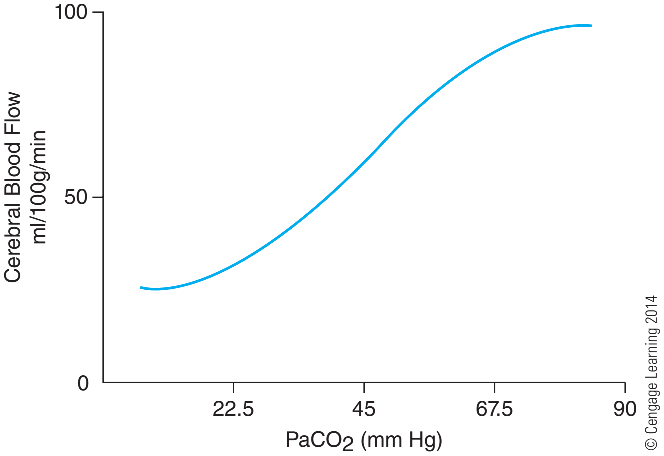

FIGURE 15-3 Cerebral blood flow in relation to the arterial carbon dioxide tension. There is no significant reduction in cerebral blood flow below a $PaCO_2$ of 26 mm Hg.

lower cerebral blood flow and intracranial pressure. The $PaCO_2$ may be titrated to a level as low as 26 torr during the first 24 hours of mechanical ventilation. Hyperventilation should not cause the $PaCO_2$ level to go below 26 mm Hg (3.5 kPa) because there is no additional beneficial effect on the ICP with a $PaCO_2$ below 26 mm Hg (Figure 15-3) (trauma.org[2], 2011).

Arterial oxygen saturation should be monitored, and the PaO_2 and SaO_2 should be kept above 60 mm Hg and 90%, respectively (Brain Trauma Foundation, 2011).

✓ To maintain minimal cerebral oxygenation, the PaO_2 and SaO_2 should be kept above 60 mm Hg and 90%, respectively.

SUMMARY

Mechanical ventilation is a common procedure in the ICU and other nontraditional settings. ALI/ARDS, VAP, HIE, and TBI are some critical care issues that are frequently encountered by respiratory therapists. This chapter provides a review of these issues. Management of critically ill patients can be complicated and tedious. Weaning from mechanical ventilation is not possible until the patients are free from ALI/ARDS and VAP. For the best patient care, readers are encouraged to keep abreast of current ventilator-related critical care topics.

Self-Assessment Questions

1. A PaO_2/F_IO_2 of ≤200 mm Hg is a characteristic of patients with:

   A. ventilator-associated pneumonia.
   B. acute lung injury.
   C. hypoxic-ischemic encephalopathy.
   D. acute respiratory distress syndrome.

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2. Bilateral infiltrates on the chest radiograph and elevated pulmonary capillary wedge pressure (e.g., 22 mm Hg) are signs of:

   A. cardiogenic pulmonary edema.
   B. noncardiogenic pulmonary edema.
   C. ARDS.
   D. ALI.

3. In order to reduce the risk of lung injuries during mechanical ventilation, the ARDSNet recommends that the plateau pressure be kept below:

   A. 15 cm H_2O.
   B. 20 cm H_2O.
   C. 30 cm H_2O.
   D. 40 cm H_2O.

4. The physician wants to implement permissive hypercapnia for a mechanically ventilated patient. The therapist should reduce the _______ setting in the range of _______.

   A. tidal volume, 8–10 mL/Kg
   B. tidal volume, 4–7 mL/Kg
   C. peak inspiratory pressure, 10–20 cm H_2O
   D. peak inspiratory pressure, 20–30 cm H_2O

5. The ARDSNet initial settings for patients with ARDS include all of the following *except*:

   A. volume-controlled ventilation.
   B. assist/control mode.
   C. maintenance of SpO_2 between 88 and 95%.
   D. use of V_T as low as 7 mL/Kg to maintain $P_{PLAT} < 30$ cm H_2O.

6. The decremental recruitment maneuver is done to obtain a patient's:

   A. optimal PEEP.
   B. static compliance.
   C. dynamic compliance.
   D. airflow resistance

7. A patient has a score of 8 using the modified clinical pulmonary infection score chart. This means that the patient is likely to require:

   A. mechanical ventilation.
   B. antibiotics.
   C. saline lavage and suctioning of the ET tube.
   D. pulmonary isolation.

8. The RT department is implementing a policy to reduce the incidence of ventilator-associated pneumonia. Based on research findings, which of the following methods should *not* be included in the policy?

   A. Reduce the use of sedatives.
   B. Elevate head of bed to 30- to 45-degree angle.
   C. Change ventilator circuit every 24 hours.
   D. Avoid intubation and use noninvasive ventilation.

9. A patient's calculated cerebral perfusion pressure (CPP) is 60 mm Hg (normal 70 to 80 mm Hg). What can be done to increase the patient's CPP?

   A. Increase the mean arterial pressure.
   B. Decrease the intracranial pressure.
   C. Increase the intracranial pressure.
   D. A and B only.

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10. A patient in the emergency department who was involved in a motor vehicle crash has a Glasgow Coma Scale score of 7. This means that the patient has:

    A. no brain injury.
    B. severe brain injury.
    C. moderate brain injury.
    D. coma.

11. Based on a study published in trauma.org, the intracranial pressure may be lowered by ________ a patient to a $PaCO_2$ level of ________ during the first 24 hours of mechanical ventilation.

    A. hyperventilating, 26 mm Hg
    B. hypoventilating, 55 mm Hg
    C. hyperventilating, 55 mm Hg
    D. hypoventilating, 26 mm Hg

Answers to Self-Assessment Questions

1. D.
2. A.
3. C.
4. B.
5. D.
6. A.
7. B.
8. C.
9. D.
10. B.
11. A.

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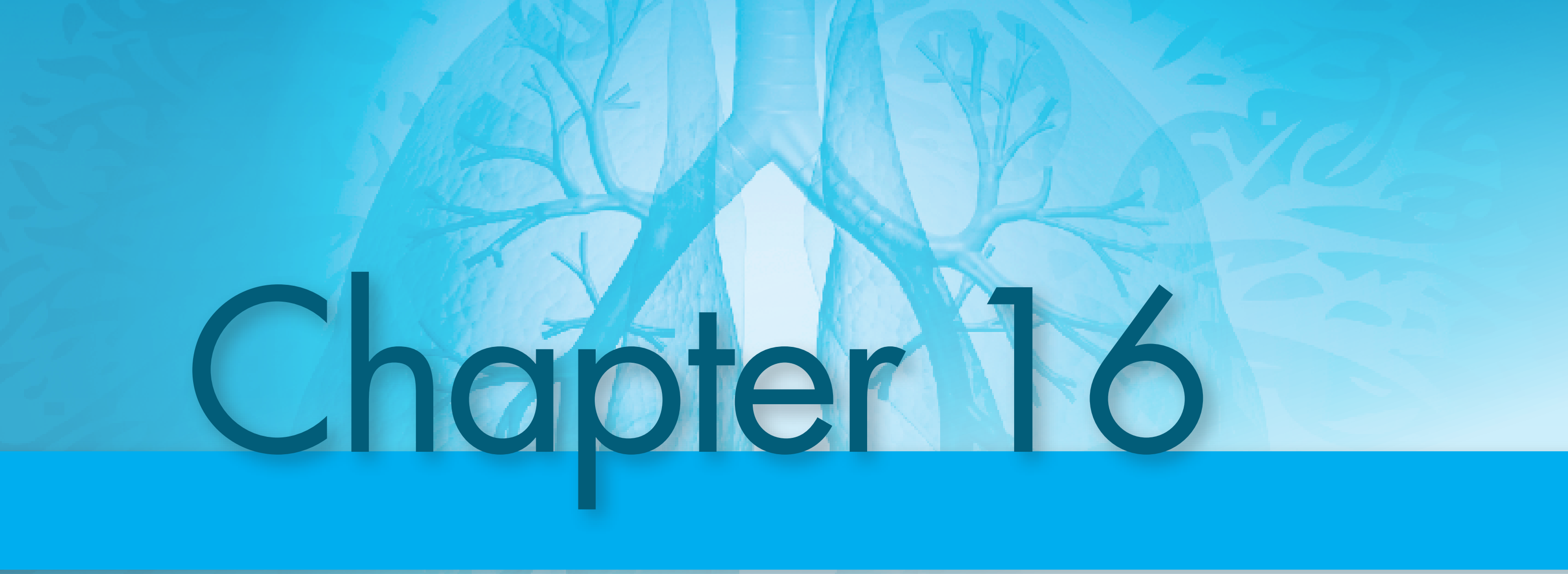

Chapter 16

Weaning from Mechanical Ventilation

David W. Chang
James H. Hiers

Outline

- Introduction
- Definition of Weaning Success and Failure
  - *Weaning Success*
  - *Weaning in Progress*
  - *Weaning Failure*
- Patient Condition Prior to Weaning
- Weaning Criteria
  - *Ventilatory Criteria*
  - *Oxygenation Criteria*
  - *Pulmonary Reserve*
  - *Pulmonary Measurements*
- Rapid Shallow Breathing Index (RSBI)
- Weaning Procedure
  - *Spontaneous Breathing Trial*
  - *Failure of SBT*
  - *Pressure Support Ventilation*
  - *Other Modes of Partial Ventilatory Support*
- Weaning Protocol
- Signs of Weaning Failure
- Causes of Weaning Failure
  - *Increase of Airflow Resistance*
  - *Decrease of Compliance*
  - *Respiratory Muscle Fatigue*
- Terminal Weaning
  - *Prior to Withdrawal*
  - *Withdrawal*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

Key Terms

- pressure support ventilation (PSV)
- rapid shallow breathing index (RSBI)
- spontaneous breathing trial (SBT)
- synchronized intermittent mandatory ventilation (SIMV)
- weaning failure
- weaning in progress
- weaning success

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Define weaning success, weaning in progress, and weaning failure.
- List, describe, or calculate the weaning criteria for assessing ventilation, oxygenation, pulmonary reserve, and pulmonary measurements.
- Calculate and interpret the rapid shallow breathing index (RSBI).
- Describe the following weaning procedures: spontaneous breathing trial, SIMV, pressure support ventilation, and other partial ventilator support.
- List the indicators of weaning failure.
- List the causes of weaning failure.
- Differentiate withholding and withdrawing of mechanical ventilation.

INTRODUCTION

Weaning is the process of withdrawing mechanical ventilatory support and transferring the work of breathing from the ventilator to the patient. In most cases, weaning may be accomplished rapidly from full ventilatory support to unassisted spontaneous breathing.

Many patients can tolerate an abrupt termination of ventilatory support; this would include those who have been on the ventilator for a relatively short time (usually no more than 1 to 2 days) and who have also regained normal cardiopulmonary function. Examples include patients recovering from postanesthesia, drug overdose, and status asthmaticus.

For other patients, successful weaning requires a more gradual withdrawal of mechanical ventilatory support. Generally, the longer the patient has been on mechanical ventilation, the more gradual the weaning process should be. The process of gradually reducing mechanical ventilatory support must be individualized and the weaning process may take from days to weeks or even months. Indeed, some patients become ventilator-dependent and may not be able to maintain adequate ventilation without mechanical assistance. Examples of these patients include high cervical spine injury, traumatic brain injury, and some neuromuscular diseases.

DEFINITION OF WEANING SUCCESS AND FAILURE

✓ The process of gradually reducing mechanical ventilatory support must be individualized for each patient.

The ability to breathe spontaneously is the criterion to gauge the success or failure of weaning attempts. Weaning success means that a patient is able to maintain spontaneous breathing for a prescribed period of time. This usually leads to termination of mechanical ventilation. Weaning failure generally means that a patient is returned to mechanical ventilation after a period of unsustained spontaneous breathing.

Weaning Success

weaning success: Absence of ventilatory support 48 hours following the extubation.

Weaning success is defined as absence of ventilatory support 48 hours following the extubation (Boles et al., 2007). While the spontaneous breaths are unassisted by mechanical ventilation, supplemental oxygen, bronchodilators, pressure support ventilation, or continuous positive airway pressure may be used to support and maintain adequate spontaneous ventilation and oxygenation.

Success Rate. Not all patients can be weaned from mechanical ventilation successfully. One retrospective review of six studies reports that a combined 79% of 2,486 patients passed the initial SBT and the weaning success rate was 68.8%. Thirteen percent of the patients who had passed the initial SBT required reintubation (Boles et al., 2007). The duration needed to wean a patient from mechanical ventilation may also vary greatly. One study shows 15% of mechanically ventilated patients required more than 7 days to be weaned successfully (Nett et al., 1984).

✓ Since medical patients often have coexisting problems, they usually take more time to complete the weaning process than surgical patients.

The success rate of weaning attempts is partly dependent on the patient population. It is generally more difficult to predict the weaning outcome of patients with medical conditions. Since these patients often have coexisting or chronic medical problems, they usually take more time to complete the weaning process than surgical patients (Yang et al., 1991).

Weaning in Progress

weaning in progress: An intermediate category (between weaning success and weaning failure) for patients who are extubated but continue to receive ventilatory support by noninvasive ventilation (NIV).

Weaning in progress is an intermediate category (between weaning success and weaning failure) for patients who are extubated but continue to receive ventilatory support by noninvasive ventilation (NIV) (Boles et al., 2007). NIV is defined as mechanical ventilation without an artificial airway. With different NIV interfaces (e.g., facial mask, nasal mask) NIV may be used to support a patient's ventilatory requirement following extubation. Use of NIV allows early weaning attempts and minimizes complications associated with prolonged mechanical ventilation and artificial airway.

✓ Weaning failure is defined as either the failure of spontaneous breathing trial (SBT) or the need for reintubation within 48 hours following extubation.

Weaning Failure

weaning failure: Failure of spontaneous breathing trial (SBT) or the need for reintubation within 48 hours following extubation.

Weaning failure is more difficult to define than weaning success. This is because whenever a patient is placed back on the ventilator, the weaning attempt has failed in one form or another. In most studies, **weaning failure** is defined as either the failure of spontaneous breathing trial (SBT) or the need for reintubation within 48 hours following extubation. Patients who fail the SBT often exhibit the following clinical signs: tachypnea, tachycardia, hypertension, hypotension, hypoxemia, acidosis, or arrhythmias. Physical signs of SBT failure may include agitation, distress, diminished mental status, diaphoresis, and increased work of breathing (Boles et al., 2007).

✓ Patients who fail the SBT often exhibit the following clinical signs: tachypnea, tachycardia, hypertension, hypotension, hypoxemia, acidosis, or arrhythmias.

Failure to remain extubated and the need for reintubation impose another problem in the overall weaning process. Weaning becomes more challenging in patients with an earlier failed attempt. Excessive secretions, hypercapnia, prolonged mechanical

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ventilation (>72 hours), and upper airway disorders are some key factors that affect the weaning outcome (Boles et al., 2007).

Impact of Weaning Failure. It is obvious that patients who require prolonged ventilator support have a severe impact on the use of health care resources, including financial, physical, and human resources. In addition, the longer a patient stays on the ventilator, emotional and psychological pains also take a toll. For these reasons, different strategies have been devised to enhance the weaning process. These strategies are designed to minimize the use of limited resources, and to keep the patients from the uncomfortable and sometimes painful diagnostic and therapeutic procedures associated with mechanical ventilation.

PATIENT CONDITION PRIOR TO WEANING

✓ Before weaning, the patient should have recovered from the acute phase of the disease leading to mechanical ventilation and be able to assume adequate spontaneous breathing.

Perhaps the first consideration before any weaning attempt is to assess the patient's overall clinical status. Two important questions pertaining to the patient's clinical condition are (1) Has the patient significantly recovered from the acute phase of the disease or injury that prompted the need for mechanical ventilation? (2) Are there other clinical conditions that may interfere with the patient's ability to sustain the work of spontaneous breathing?

Assessment of the patient's overall clinical condition should include an evaluation of the clinical conditions in Table 16-1. Depending on the severity of these clinical conditions, they should be corrected or normalized prior to a weaning attempt.

TABLE 16-1 Conditions That May Hinder a Successful Weaning Outcome

Condition	Example
Patient/pathophysiologic	Fever Infection Renal failure Sepsis Sleep deprivation
Cardiac/circulatory	Arrhythmias Blood pressure (high or low) Cardiac output (high or low) Fluid imbalance Anemia Dysfunctional hemoglobins
Dietary/acid-base/electrolytes	Nutritional or caloric deficit Acid-base imbalance Electrolytes imbalance

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WEANING CRITERIA

Weaning criteria are used to evaluate the readiness of a patient for a weaning trial and the likelihood of weaning success. Weaning is more likely to succeed if a patient meets most of the criteria.

Both subjective and objective measurements have been developed to determine a patient's readiness for weaning. The common weaning criteria are summarized in Table 16-2.

Ventilatory Criteria

The ventilatory status of a patient may be used to evaluate the readiness and outcome of weaning attempts. Weaning success will be more likely if the patient can

TABLE 16-2 Common Weaning Criteria

Category	Example	Note
Clinical criteria	Resolution of acute phase of disease Adequate cough Absence of excessive secretions Cardiovascular and hemodynamic stability	
Ventilatory criteria	Spontaneous breathing trial	Tolerates 20 to 30 min
	$PaCO_2$	<50 mm Hg with normal pH
	Vital capacity	>10 mL/kg
	Spontaneous V_T	>5 mL/kg
	Spontaneous f	<35/min
	f/V_T	<100 breaths/min/L*
	Minute ventilation	<10 L with satisfactory ABG
Oxygenation criteria	PaO_2 without PEEP	>60 mm Hg at F_IO_2 up to 0.4
	PaO_2 with PEEP (<8 cm H_2O)	>100 mm Hg at F_IO_2 up to 0.4
	SaO_2	>90% at F_IO_2 up to 0.4
	PaO_2/F_IO_2 (P/F)	≥150 mm Hg
	Q_S/Q_T	<20%
	$P(A\text{-}a)O_2$	<350 mm Hg at F_IO_2 of 1.0
Pulmonary reserve	Vital capacity	>10 mL/kg
	Max. insp. pressure	> −30 cm H_2O in 20 sec
Pulmonary measurements	Static compliance	>30 mL/cm H_2O
	Airway resistance	Stable or improving
	V_D/V_T	<60% while intubated

**rounded from 105 breaths/min/L.*

(Data from Boles et al., 2007; Burton et al., 1997; Caruso et al., 1999; Girault et al., 1994; Jabour et al., 1991; MacIntyre, 2004; Tobin et al., 1990; Yang et al., 1991.)

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sustain an adequate ventilation. The generally accepted ventilatory weaning criteria include a PaCO$_2$ of less than 50 mm Hg with normal pH, a vital capacity of greater than 10 mL/kg, a spontaneous V$_T$ of greater than 5 mL/kg, a spontaneous frequency of less than 35/min, an f/V$_T$ of less than 100 breaths/min/L, and a minute ventilation of less than 10 L with satisfactory blood gases.

✓ The PaCO$_2$ should be between 35 and 45 mm Hg and the pH between 7.35 and 7.45 (for COPD patients, the PaCO$_2$ may be around 50 mm Hg with a pH near 7.35).

PaCO$_2$. The partial pressure of carbon dioxide in the arterial blood (PaCO$_2$) is a reliable indicator of the patient's ventilatory status. Weaning from mechanical ventilation should be attempted only when the PaCO$_2$ is less than 50 mm Hg with a compensated pH (non-COPD patient).

In patients with normal lung functions, the PaCO$_2$ should be within the normal range of 35–45 mm Hg and the pH should be between 7.35 and 7.45. However, in patients with COPD, the acceptable PaCO$_2$ may be slightly higher and the pH slightly lower, depending on the patient's baseline normal values prior to mechanical ventilation (Millbern et al., 1978).

✓ The vital capacity and spontaneous tidal volume that correlate with successful weaning are 10 mL/kg and 5 mL/kg, respectively.

Vital Capacity and Spontaneous Tidal Volume. The mechanical condition of the lungs may be evaluated by measuring the vital capacity and spontaneous tidal volume. It is generally accepted that the minimal vital capacity and spontaneous tidal volume consistent with successful weaning are 10 mL/kg and 5 mL/kg, respectively (Pierson, 1982, 1983; Tahvanainen et al., 1983). The results of 11 studies indicate that spontaneous tidal volume averaged 368 mL in weaned patients but only averaged 277 mL in nonweaned patients (Jabour et al., 1991).

If the patient has been receiving full ventilatory support, it is advisable to allow the patient to breathe spontaneously for 3 min under close observation prior to measuring the vital capacity and spontaneous tidal volume. An equilibration period is needed to obtain the spontaneous effort based on the patient's actual respiratory requirement.

✓ The vital capacity maneuver is effort-dependent. Proper teaching and coaching are required for accurate and valid measurements.

Unlike spontaneous tidal volume, vital capacity requires active patient effort and cooperation. Vital capacity measures the maximal amount of volume that the patient can expire following a maximal inspiration. For this reason, its validity is effort-dependent, and proper teaching and coaching are required for accurate measurements. Poor effort or inability to follow commands may result in lower than actual vital capacity measurement.

✓ Rapid shallow breathing increases deadspace ventilation and is highly ineffective for gas exchange during spontaneous breathing.

Spontaneous Frequency. For a successful weaning outcome, the spontaneous frequency should be less than 35/min while the corresponding PaCO$_2$ should be less than 50 mm Hg (Pierson, 1983; Tahvanainen et al., 1983). A frequency of greater than 35/min is associated with rapid shallow breathing. This breathing pattern increases deadspace ventilation and is highly ineffective for gas exchange during spontaneous breathing. A moderate to significant increase in spontaneous frequency after discontinuation of mechanical ventilation is a sign of impending weaning failure (Jabour et al., 1991).

As with the spontaneous tidal volume measurement, the patient should be allowed to breathe spontaneously for 3 min prior to measuring the spontaneous frequency. This allows the patient ample time to normalize the breathing pattern, and thus is more reflective of the patient's response to the respiratory requirement.

An f/V_T index of 100 breaths/min/L correlates with weaning success.

f/V_T. The f/V_T index reflects the degree of rapid shallow breathing. In patients who are breathing rapidly with small tidal volumes, the V_D/V_T ratio and f/V_T index would be increased. An f/V_T index of 100 breaths/min/L correlates with weaning success.

For a successful weaning outcome, the patient's minute ventilation should be *less* than 10 L/min with acceptable blood gases.

Minute Ventilation. The patient's minute ventilation (either spontaneous or assisted) should be *less* than 10 L/min for a successful weaning outcome (assuming the corresponding $PaCO_2$ is normal). A high minute ventilation requirement (>10 L) to normalize the $PaCO_2$ implies that the work of spontaneous breathing is excessive. The patient is unlikely to be able to sustain the increased work of breathing once the weaning process begins.

An excessive minute ventilation requirement may result from increased carbon dioxide production secondary to an increased metabolic rate, an increase in alveolar deadspace, or metabolic acidosis. Causes for increased carbon dioxide production include extensive burn injuries, an elevated body temperature, and sometimes overfeeding, especially with excessive carbohydrate supplements. Alveolar deadspace will be increased if the alveolar ventilation exceeds the alveolar perfusion ($V/Q > 0.8$). This condition of deadspace ventilation may occur when (1) the alveoli are overventilated as in hyperinflation of the lungs (e.g., emphysema) and (2) the pulmonary circulation is underperfused (e.g., pulmonary embolism, decreased cardiac output).

Oxygenation Criteria

The oxygenation status of a patient may be used to evaluate the readiness and outcome of weaning attempts. Weaning success will be more likely if the patient is adequately oxygenated while receiving partial or no ventilatory support before or during the weaning process. The generally accepted oxygenation weaning criteria include a PaO_2 of greater than 60 mm Hg (or $SaO_2 > 90\%$) on an F_IO_2 of 0.40 or less (Barnes, 1994), a PaO_2/F_IO_2 index greater than 150 mm Hg, an intrapulmonary shunt (Q_S/Q_T) of less than 20%, and an alveolar-arterial oxygen tension gradient ($P_{(A\text{-}a)}O_2$) less than 350 mm Hg at an F_IO_2 of 100% (Boles et al., 2007; Feeley et al., 1975; Girault et al., 1994).

For a successful weaning outcome, the PaO_2 and SaO_2 should be >60 mm Hg and >90%, respectively on F_IO_2 < 40%.

PaO_2 and SaO_2. A PaO_2 of 60 mm Hg corresponds to an SaO_2 of about 90%. It is essential to note that in patients with anemia or increased level of dysfunctional hemoglobins (carboxyhemoglobin), the PaO_2 and SpO_2 (pulse oximetry) do not reflect the true oxygenation status of the patient. In those instances, the arterial oxygen content (CaO_2) and arterial oxygen saturation (SaO_2) should be measured and used for clinical decisions.

If pulse oximetry is used to monitor a patient's oxygenation status, the pulse oximetry O_2 saturation (SpO_2) should be kept in the mid-90s for allowance of machine inaccuracies, because SpO_2 readings in critical care are accurate to within 2% to 4% of the SaO_2 (Malley, 1990).

For a successful weaning outcome, the PaO_2/F_IO_2 should be ≥150 mm Hg.

PaO_2/F_IO_2. The arterial oxygen tension to inspired oxygen concentration (PaO_2/F_IO_2) or P/F index is a simplified method for estimating the degree of intrapulmonary shunt. A PaO_2/F_IO_2 of ≥150 mm Hg suggests acceptable physiologic shunt

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and compatible to successful weaning trial (Girault et al., 1994; Grap et al., 2003; MacIntyre et al., 2001).

QS/QT. The physiologic shunt to total perfusion (Q_S/Q_T) ratio is used to estimate how much pulmonary perfusion is wasted. Shunted pulmonary perfusion cannot take part in gas exchange due to mismatch of ventilation (e.g., atelectasis). The Q_S/Q_T ratio can be calculated using the classic physiologic shunt equation:

✔ See Appendix 1 for example.

$$\frac{Q_S}{Q_T} = \frac{(CcO_2 - CaO_2)}{(CcO_2 - C\bar{v}O_2)}$$

Q_S/Q_T: Shunt percent in %

CcO_2: End-capillary oxygen content in vol%

CaO_2: Arterial oxygen content in vol%

$C\bar{v}O_2$: Mixed venous oxygen content in vol%

✔ For a successful weaning outcome, the Q_S/Q_T should be $<$20%.

In clinical settings, a calculated physiologic shunt of 10% or less is considered normal. Shunt of 10% to 20% indicates mild physiologic shunt, and shunt of 20% to 30% shows significant physiologic shunt. Greater than 30% shunt reflects critical and severe shunt (Malley, 1990; Shapiro et al., 1994).

Since physiologic shunt in mechanical ventilation is usually intrapulmonary in origin (inadequate ventilation in relation to pulmonary perfusion), weaning failure becomes likely when spontaneous ventilation cannot keep up with the pulmonary perfusion. For this reason, significant and severe intrapulmonary shunt ($Q_S/Q_T > 20\%$) should be corrected before any weaning attempt.

$P_{(A-a)}O_2$. The alveolar-arterial oxygen tension gradient ($P_{(A-a)}O_2$) is used to estimate the degree of hypoxemia and the degree of physiologic shunt. The $P_{(A-a)}O_2$ gradient may be obtained by subtracting the measured PaO_2 from the calculated P_AO_2 value. This gradient is directly related to the degree of hypoxemia or shunt (a larger gradient reflects more severe hypoxemia or shunt).

✔ See Appendix 1 for example of P(A-a)O_2.

The alveolar-arterial oxygen tension gradient ($P_{(A-a)}O_2$) can be calculated as follows:

$$P_{(A-a)}O_2 = P_AO_2 - PaO_2$$

$P_{(A-a)}O_2$: Alveolar-arterial oxygen tension gradient in mm Hg

P_AO_2: Alveolar oxygen tension in mm Hg

PaO_2: Arterial oxygen tension in mm Hg

✔ On 100% oxygen, every 50 mm Hg difference in $P_{(A-a)}O_2$ approximates 2% physiologic shunt.

On room air, the $P_{(A-a)}O_2$ should be less than 4 mm Hg for every 10 years in age. For example, the $P_{(A-a)}O_2$ should be less than 24 mm Hg for a 60-year-old patient. On 100% oxygen, every 50 mm Hg difference in $P_{(A-a)}O_2$ approximates 2% physiologic shunt (Barnes, 1994; Burton et al., 1997; Shapiro et al., 1994).

✔ For a successful weaning outcome, the $P_{(A-a)}O_2$ should be $<$350 mm Hg while on 100% O_2.

In mechanical ventilation, $P_{(A-a)}O_2$ of less than 350 mm Hg while on 100% oxygen suggests a likelihood of weaning success. $P_{(A-a)}O_2$ of 350 mm Hg while on 100% oxygen approximates 14% shunt and values of greater than 350 mm Hg may

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hinder the weaning process. Any large $P_{(A\text{-}a)}O_2$ gradient (>350 mm Hg) should be corrected prior to the weaning trial.

Pulmonary Reserve

A patient's pulmonary reserve may be assessed by measuring the vital capacity (VC) and maximum inspiratory pressure (MIP). The VC and MIP maneuvers require active patient cooperation, and therefore these two measurements are effort-dependent. Proper explanation, vigorous coaching, and allowance of an equilibration period to stimulate active respiratory drive are the prerequisites for valid and meaningful measurements.

✓ For a successful weaning outcome, the patient should have a VC >10 mL/Kg.

Vital Capacity. The vital capacity (VC) reflects a patient's pulmonary reserve as it includes three lung volumes: inspiratory reserve volume, tidal volume, and expiratory reserve volume. VC measures the maximum amount of lung volume that the patient can exhale following maximal inspiration. Typically the patient is instructed to breathe in as deeply as possible and exhale all the air into a spirometer. Unlike the forced vital capacity obtained in the pulmonary function lab, this VC maneuver does not require forceful exhalation. For successful weaning, the patient should have a VC of greater than 10 mL/kg (Boles et al., 2007; Pierson, 1982, 1983; Tahvanainen et al., 1983).

Maximum Inspiratory Pressure. The maximum inspiratory pressure (also called negative inspiratory force) is the amount of negative pressure that the patient can generate in 20 sec when inspiring against an occluded measuring device (negative pressure manometer) (Marini et al., 1986). If the patient is alert, explain the procedure and encourage the patient to attempt to inspire as forcibly as possible. In some mechanically ventilated patients, a waiting period without assisted ventilation may be needed to induce mild hypoxia and hypercapnia for the best inspiratory efforts. In addition, the duration of airway occlusion is an important factor in determining the accuracy (individual therapists) and reliability (between therapists) of the MIP measurements (Soo Hoo, 2002).

✓ Weaning will likely be successful if the patient can generate an MIP of at least −30 cm H_2O.

The MIP is considered a measure of ventilatory muscle strength, and weaning will likely be successful if the patient can generate an MIP of at least −30 cm H_2O. The results of 11 studies indicate that the MIP *averaged* −37 cm H_2O for weaned patients versus only −30 cm H_2O for nonweaned patients (Jabour et al., 1991).

Pulmonary Measurements

Static compliance, airway resistance, and deadspace to tidal volume (V_D/V_T) ratio are three measurements that are not dependent on a patient's cooperation or effort. They are used to indicate the amount of pulmonary workload that is needed to support spontaneous ventilation. In general, low compliance, high airway resistance, and high V_D/V_T ratio all contribute to an increased workload. When these undesirable conditions reach the patient's threshold, they may hinder the weaning process and outcome.

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For a successful weaning outcome, the compliance should be >30 mL/cm H_2O.

Static Compliance. The static lung compliance is measured by dividing the patient's tidal volume (measured at the airway opening) by the difference in the plateau pressure and the PEEP. The lower the compliance, the greater the work of breathing will be. The minimal compliance value consistent with successful weaning is 30 mL/cm H_2O or greater (Hess et al., 1991).

The static lung compliance may be calculated as follows:

See Appendix 1 for example.

$$C_{ST} = \frac{\Delta V}{\Delta P}$$

C_{ST}: Static lung compliance in mL/cm H_2O
ΔV: Corrected tidal volume in mL
ΔP: Pressure change ($P_{PLAT} - PEEP$) in cm H_2O

Airway Resistance. The airway resistance can be estimated by dividing the difference in the peak inspiratory pressure and the plateau pressure (H_2O) by the *constant* inspiratory flow (L/sec). The normal range for airway resistance is 0.6–2.4 cm H_2O/L/sec (Burton et al., 1991) and higher for ventilator patients because of the associated pathological conditions (e.g., bronchospasm) and tubing resistance (e.g., endotracheal tube, ventilator circuit).

Although no critical weaning value for airway resistance has been established for mechanical ventilation, the work of breathing is directly related to the degree of airway resistance. The endotracheal (ET) tube contributes significantly to the airway resistance. The effect of resistance through the tube can be minimized by ensuring that the ET tube is not kinked or the suction catheter of a continuous suction system is not protruding into the tube. Since retained secretions and bronchospasm contribute to the airway resistance, the patient's airways and lungs should be suctioned as needed. Use of bronchodilators may be helpful to reduce the airway resistance in conditions of reversible bronchospasm.

pressure support ventilation (PSV): A mode of ventilation in which the patient's spontaneous tidal volume is augmented by the application of a preset pressure plateau to the patient's airway during the inspiratory phase of a spontaneous breath.

Pressure support ventilation (PSV) has been used extensively to reduce the elastic and nonelastic airflow resistance and to augment the spontaneous tidal volume. The resulting spontaneous tidal volume is directly related to the pressure support level. PSV may be used in the spontaneous breathing mode or in conjunction with other modes of ventilation that include spontaneous breathing (e.g., SIMV). PSV cannot be used in modes of ventilation that do not allow spontaneous breathing.

Since airflow resistance is highly variable among patients and at different stages of mechanical ventilation, the pressure support level must be monitored and adjusted accordingly. The initial PSV level should be titrated until a desired spontaneous tidal volume is reach (e.g., 10 to 15 mL/kg) or until the spontaneous frequency decreases to a target value (e.g., ≤25/min) (MacIntyre, 1987).

The initial PSV level should be titrated until a desired spontaneous tidal volume is reach (e.g., 10 to 15 mL/kg) or until the spontaneous frequency decreases to a target value (e.g., ≤25/min).

Deadspace/Tidal Volume (V_D/V_T) Ratio. The deadspace to tidal volume (V_D/V_T) ratio indicates the amount of each breath that is "wasted" or not being perfused by pulmonary circulation. The higher the V_D/V_T ratio, the greater the minute volume demand will be. The V_D/V_T ratio can be calculated as the partial pressure of arterial carbon dioxide minus the mean partial pressure of the carbon dioxide in the exhaled air divided by the arterial blood carbon dioxide tension. For a successful weaning outcome, the V_D/V_T ratio should be 60% or less (Fitzgerald et al., 1976).

✓ For a successful weaning outcome, the V_D/V_T ratio should be <60%.

The deadspace to tidal volume (V_D/V_T) ratio can be calculated as follows:

$$\frac{V_D}{V_T} = \frac{(PaCO_2 - P_{\bar{E}}CO_2)}{PaCO_2}$$

✓ See Appendix 1 for example.

V_D/V_T: Deadspace to tidal volume ratio in %

$PaCO_2$: Arterial carbon dioxide tension in mm Hg

$P_{\bar{E}}CO_2$: Mixed expired carbon dioxide tension in mm Hg

RAPID SHALLOW BREATHING INDEX (RSBI)

rapid shallow breathing index (RSBI): The RSBI (f/V_T index) is calculated by dividing the spontaneous breathing frequency (breaths/min) by the average spontaneous V_T (L). Absence of rapid shallow breathing, as defined by an f/V_T ratio of less than 100* breaths/min/L, is an accurate predictor of weaning success. (*rounded from 105 breaths/min/L.)

Failure of weaning may be related to the development of a spontaneous breathing pattern that is rapid (high frequency) and shallow (low tidal volume). The **rapid shallow breathing index (RSBI)** or f/V_T index has been used to evaluate the effectively of the spontaneous breathing pattern (Jacob et al., 1997; Tobin et al., 1986; Vassilakopoulos et al., 1998; Yang et al., 1991).

Rapid shallow breathing is quantified as the f (number of breaths per minute) divided by the V_T in liters, and this breathing pattern induces inefficient, deadspace ventilation. When the RSBI or f/V_T index is greater than 100 breaths/min/L (rounded from 105 breaths/min/L), it correlates with weaning failure. On the other hand, absence of rapid shallow breathing (f/V_T ratio <100 breaths/min/L), is an accurate predictor of weaning success (Yang et al., 1991).

✓ Rapid shallow breathing induces inefficient, deadspace ventilation.

To measure the f/V_T index, the patient is taken off the ventilator and allowed to breathe spontaneously for 3 min or until a stable breathing pattern has been established. The minute expired volume (V_E) and spontaneous frequency (f) are measured. The average V_T is calculated by dividing the V_E by f. The f/V_T is calculated by dividing the f (breaths/min) by the average V_T (L). The procedure for measuring and calculating the f/V_T is outlined in Table 16-3.

TABLE 16-3 Procedures to Obtain the f/V_T Ratio

Procedure
1. Allow the patient at least 3 min to stabilize the spontaneous breathing pattern (ventilator frequency must be off and PSV should not be used if tolerated by patient)
2. Measure expired volume and respiratory frequency for one min
3. Divide minute volume by frequency (f) to obtain an average tidal volume (V_T) in liter
4. Divide f by V_T to obtain f/V_T index (breaths/min/L)

Note: See Appendix 1 for example.

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WEANING PROCEDURE

spontaneous breathing trial (SBT): An evaluation of a patient's readiness for weaning from mechanical ventilation and extubation. Spontaneous breathing may be augmented with low-level (≤8 cm H_2O) of pressure support, CPAP, or automatic tube compensation (ATC). SBT may last up to 30 minutes.

synchronized intermittent mandatory ventilation (SIMV): A mode of ventilation that permits spontaneous breaths between ventilator breaths. The ventilator breaths are synchronized (mandatory breaths that may come slightly sooner or later) to coincide with the patient's inspiratory efforts.

The **spontaneous breathing trial (SBT)** is the major diagnostic test to determine if patients can be successfully extubated and weaned from mechanical ventilation. Low level pressure support (PS), continuous positive airway pressure (CPAP), or automatic tube compensation (ATC) may be used along with SBT to augment a patient's spontaneous breathing efforts (Keenan, 2002). Based on the results of the sixth International Consensus Conference on Intensive Care Medicine, **synchronized intermittent mandatory ventilation (SIMV)** should be avoided as a stand-alone weaning modality (Boles et al., 2007). However, SIMV remains an effective tool in providing partial ventilatory support during continuous mechanical ventilation.

Spontaneous Breathing Trial

Once a decision is made to proceed with weaning, the patient may be discontinued from full ventilatory support and placed on a spontaneous breathing mode via the ventilator or T-tube (Brigg's adaptor) for up to 30 minutes. Oxygen and low level pressure support may be used to supplement oxygenation and augment spontaneous breathing.

The criteria for passing an SBT include normal respiratory pattern (i.e., absence of rapid shallow breathing), adequate gas exchange, and hemodynamic stability. The results of six studies show that only 13% of patients who successfully passed the SBT and were extubated required reintubation (Boles et al., 2007).

There is no difference in terms of successful SBT among patients undergoing stand-alone SBT, SBT with low level of pressure support, and SBT with CPAP or automatic tube compensation (Boles et al., 2007). Since patients who fail the SBT do so within the first 20 to 30 minutes of SBT, there is no need to use an extended SBT (e.g.,120-min trial) (Yang et al., 1991; Perren et al., 2002). Table 16-4 describes the procedure for the spontaneous breathing trial and other partial ventilatory support procedures. Note that SIMV is used to provide partial ventilatory support and it is not recommended as a stand-alone weaning modality.

Failure of SBT

Patients who fail the SBT often do so within the first 20 to 30 minutes of the trial. They also exhibit the following clinical signs and symptoms: agitation and anxiety, diminished mental status, diaphoresis, cyanosis, and evidence of increased work of breathing (Boles et al., 2007). Clinical data that correlate with failure of SBT are summarized in Table 16-5.

Pressure Support Ventilation

Pressure support ventilation (PSV) or similar adjuncts (e.g., proportional pressure support, volume-assured pressure support) may be applied during weaning. PSV

TABLE 16-4 Spontaneous Breathing Trial (SBT) and Partial Ventilatory Support Procedures

Procedure	Steps
SBT	(1) May use T-tube, CPAP, or automatic tube compensation; (2) Let patient breathe spontaneously for up to 30 min.; (3) May use low level pressure support (up to 8 cm H_2O for adults and 10 cm H_2O for pediatrics) to augment spontaneous breathing; (4) Assess patient; (5) If patient tolerates step (4), consider extubation when blood gases and vital signs are satisfactory. Return patient to mechanical ventilation to rest if necessary.
SIMV (not recommended as a stand-alone mode for weaning)	(1) Reduce SIMV (ventilator) frequency by 1 to 3 breaths per min; (2) Monitor SpO_2, obtain ABG as needed; (3) Reduce SIMV frequency further until a frequency of 2 to 4/min is reached. This may take only hours for patients with normal cardiopulmonary functions but days for those with abnormal functions; (4) If patient tolerates step (3), consider extubation when blood gases and vital signs are satisfactory.
PSV	(1) PSV may be used in conjunction with spontaneous breathing or SIMV mode; (2) Start PSV at a level of 5 to 15 cm H_2O (up to 40 cm H_2O) to augment spontaneous V_T until a desired V_T (10 to 15 mL/kg) or spontaneous frequency ($\leq$25/min) is reached; (3) Decrease pressure support (PS) level by 3 to 6 cm H_2O intervals until a level of close to 5 cm H_2O is reached; (4) If patient tolerates step (3), consider extubation when blood gases and vital signs are satisfactory.

(Data from Boles et al., 2007; Downs et al., 1974; Girault et al., 1999; MacIntyre, 1986; MacIntyre, 1987, Milbern et al., 1978; Nett et al., 1984; Tobin et al., 1990.)

helps to reduce the airflow resistance imposed on the patient by the endotracheal tube and ventilator circuit. Some clinicians advocate weaning with pressure support as a stand-alone mode. Regardless of the weaning approach used, it is advisable to provide full ventilatory support at night to allow the patient to rest (Barnes, 1994).

✓ Weaning with PSV is done by starting the pressure support level at 5 to 15 cm H_2O and adjusting it gradually (up to 40 cm H_2O) until a desired spontaneous V_T (10 to 15 mL/kg) or spontaneous frequency ($\leq$25/min) is obtained.

Weaning with PSV is done by starting the pressure support level at 5 to 15 cm H_2O and adjusting it gradually (up to 40 cm H_2O) until a desired spontaneous V_T (10 to 15 mL/kg) is obtained (MacIntyre, 1986, 1987). Some practitioners titrate the pressure support level until a desired spontaneous frequency is reached, typically 25/min or less. This approach is clinically relevant since an increased

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TABLE 16-5 Clinical Criteria and Thresholds Related to SBT Failure

$PaO_2 \leq 60$ mm Hg on $F_IO_2 \geq 50\%$
$SaO_2 < 90\%$ on $F_IO_2 \geq 50\%$
$PaCO_2 > 50$ mm Hg or an increase in $PaCO_2 > 8$ mm Hg from baseline of SBT
pH < 7.32 or a decrease in pH ≥ 0.07 from baseline of SBT
$f/V_T > 100$ breaths/min/L (rounded from 105)
$f > 35$ breaths/min or increase by $\geq 50\%$ from baseline of SBT
Heart rate > 140 beats/min or increase by $\geq 20\%$ from baseline of SBT
Systolic BP > 180 mm Hg or increase by $\geq 20\%$ from baseline of SBT
Systolic BP < 90 mm Hg
Presence of cardiac arrhythmias

spontaneous tidal volume corresponds with a decreased spontaneous frequency. If the patient tolerates the weaning process well, the pressure support level is gradually decreased by 3 to 6 cm H_2O increments until a level of close to 5 cm H_2O is reached. Extubation may be considered when the patient's blood gases and vital signs remain satisfactory (Tobin et al., 1990).

Automatic Tube Compensation. Automatic tube compensation (ATC) is a mode in the Evita 4 ventilator (Dräger Medical) that reduces the airflow resistance imposed by the artificial airway (endotracheal or tracheostomy tube). It allows the patient to have a breathing pattern as if breathing spontaneously without an artificial airway. This type of compensation may facilitate breathing efficacy and reduce the work of breathing throughout the weaning process.

Other Modes of Partial Ventilatory Support

SIMV can be used to alleviate the need to alternate the patient on T-tube and ventilatory support. Using SIMV to shift the work of breathing from the ventilator to the patient is accomplished by progressively reducing the mandatory SIMV frequency (usually 1 to 3 breaths per minute at each step). Arterial blood gases (or SpO_2) may be measured after 30 min or more at that setting (Tobin et al., 1990). If the pH remains near normal (above 7.30 or 7.35) (Downs et al., 1974; Milbern et al., 1978), the SIMV frequency is further reduced in steps until a frequency of 2 to 4/min is reached. The pace of SIMV weaning is dictated by the patient's clinical condition and tolerance.

✓ The pace of SIMV weaning is dictated by the patient's clinical condition and tolerance.

Sometimes SIMV and PSV are used together in patients who have failed the spontaneous breathing trial or have done poorly with SIMV or PSV alone. Under these

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circumstances, there are other modes of ventilation that provide partial ventilatory support (MacIntyre et al., 2001). These modes are volume support, volume-assured pressure support, mandatory minute ventilation, and airway pressure-release ventilation. For a more detailed discussion on these modes, refer to Chapter 4, Operating Modes of Mechanical Ventilation.

Volume support (VS) and volume-assured pressure support (VAPS) are a form of PSV that "guarantees" a preset tidal volume. In VS, the pressure support level is adjusted automatically to achieve the target tidal volume. In VAPS, it guarantees a preset tidal volume by incorporating inspiratory pressure support ventilation (PSV) with conventional volume-assisted cycles (VAV). Unlike typical PSV, VAPS assures stable tidal volume in patients with irregular breathing patterns. By decreasing the frequency, the work of breathing is shifted from the ventilator to the patient.

Mandatory minute ventilation (MMV) is a form of SIMV in which the minute ventilation is guaranteed. The ventilator adjusts the frequency automatically to achieve the target minute ventilation. By decreasing the MMV level, the patient assumes more spontaneous breathing.

Airway pressure-release ventilation (APRV) has two pressure levels: the higher airway pressure (e.g., 10 cm H_2O) and the lower release pressure (usually 0 cm H_2O). The *tidal volume* is determined by the pressure gradient between the airway pressure and the release pressure. In APRV, exhalation occurs during pressure release and inhalation occurs when the pressure returns to the airway pressure. Weaning may be done by decreasing the frequency of pressure release. When the frequency of pressure release is zero, the patient is essentially on a CPAP mode.

WEANING PROTOCOL

Weaning protocol and clinical practice guidelines for weaning are primarily used to outline the standard of care for the purpose of weaning from mechanical ventilation. In general, they often include three elements: the patient condition in which weaning may be attempted, the detailed process of weaning, and the evaluation of weaning outcomes.

There are many weaning protocols published in the literatures or developed by individual hospitals or departments. Each of them can be useful when the elements of weaning are incorporated with sound clinical reasoning and implementation. The criteria in the weaning protocol should be used as guidelines only and must not be carried out using a "cookbook" approach. Individual patient differences must also be considered since disease processes and patient characteristics are two of many variables that may affect the outcomes of a weaning protocol (Keenan, 2002). Weaning protocols range from simple to complex. Table 16-6 provides a simple weaning protocol for mechanical ventilation (Grap et al., 2003).

TABLE 16-6 Weaning Protocol for Mechanical Ventilation

Step	Criteria	Results
1	Does the patient show: • Evidence of some reversal of underlying cause for ventilatory failure? • Presence of inspiratory effort? • Hemodynamic stability? (absence of myocardial ischemia, hypotension, use of vasopressor) • Adequate oxygenation and acid-base status? ($PaO_2/F_1O_2 > 150$ mm Hg, PEEP < 8 cm H_2O and pH ≥ 7.25) • Light sedation or better? (brief eye contact to voice command)	If YES to *all five* questions, proceed to STEP 2. If NO to *any one* question, postpone weaning until next day.
2	Perform and measure rapid shallow breathing index (RSBI or f/V_T) with mandatory frequency turned off and pressure support ≤ 8 cm H_2O, PEEP ≤ 5cm H_2O, measurements taken following ≥ 3 min of spontaneous breathing. Is RSBI (f/V_T) $<$ 100 breaths/min/L?	If YES, proceed to STEP 3. If NO, postpone weaning until next day.
3	Can patient tolerate: Spontaneous breathing trial for up to 30 minutes without termination? (See termination criteria* below.)	If YES, proceed to ventilator discontinuance or evaluate for extubation. If NO, repeat weaning until next day.

(Adapted from Grap et al., 2003 with additional data from Ely et al., 2001; MacIntyre et al., 2001.)

**Termination criteria: Spontaneous frequency $>$35/min for 5 min; $SpO_2 <$90%, Heart rate $>$140/min or 120% of baseline; Systolic pressure $>$180 mm Hg or $<$90 mm Hg; Signs of anxiety or use of accessory muscles.*

SIGNS OF WEANING FAILURE

Once the weaning process has been started, the previously described weaning criteria should be monitored closely to ensure that the patient is tolerating the weaning attempt. When the mechanical ventilatory support is decreased, part of the work of breathing is shifted to the patient. The goal for the patient is to maintain the work of spontaneous breathing and adequate oxygenation. If the patient tolerates the increased work of breathing and the weaning criteria remain within acceptable limits, then the amount of ventilatory support (e.g., SIMV frequency, pressure support level) should again be decreased. This process is repeated if the patient tolerates the decrease of ventilatory support. The weaning process should be stopped if the patient shows signs of muscle fatigue or ventilatory failure.

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✓ Patients may hyperventilate due to hypoxia, pain, anxiety, or inappropriate ventilator settings. It is a grave mistake to reduce ventilatory support simply because the $PaCO_2$ shows hyperventilation.

✓ Early signs of weaning failure include: tachypnea, use of accessory muscles and paradoxical abdominal movements, dyspnea, chest pain, chest-abdomen asynchrony, and diaphoresis.

Early signs of weaning failure include tachypnea, use of accessory muscles, and paradoxical abdominal movements (Cohen et al., 1982). It is important to evaluate and apply clinical data in conjuction with the patient's clinical presentations. Patients may hyperventilate due to hypoxia, pain, anxiety, or inappropriate ventilator settings. It is a grave mistake to reduce ventilatory support simply because the $PaCO_2$ shows hyperventilation.

Other indications that the patient cannot maintain the work of breathing may include dyspnea, chest pain, chest-abdomen asynchrony, diaphoresis, and delirium (Ely et al., 2004; Jabour et al., 1991). Some specific indicators of weaning failure are listed in Table 16-7.

If the patient does not tolerate the weaning procedure, the patient should be returned to full ventilatory support and be allowed to rest. The patient should then be reassessed in order to determine the cause of weaning failure. Appropriate therapies may then be applied before attempting the weaning process again.

TABLE 16-7 Indicators of Weaning Failure

Indicators	Examples
Blood Gases	Increasing $PaCO_2$ (>50 mm Hg)
	Decreasing pH (<7.30)
	Decreasing PaO_2 (<60 mm Hg)
	Decreasing SpO_2 (<90%)
	Decreasing PaO_2/F_IO_2 (<150 mm Hg)
Vital Signs	Changing blood pressures (20 mm Hg systolic or 10 mm Hg diastolic)
	Increasing heart rate (by 20/min, or >110/min)
	Abnormal ECG (presence of arrhythmias)
Respiratory Parameters	Decreasing V_T (<250 mL)
	Increasing f (>30/min)
	Increasing f/V_T (>100 breaths/min/L)
	Decreasing MIP (<−20 cm H_2O)
	Decreasing static compliance (<30 mL/cm H_2O)
	Increasing V_D/V_T (>60%)

(Data from Burton et al., 1997; Girault et al., 1994; Jabour et al., 1991; Jubran & Tobin, 1997; MacIntyre, et al., 2001; Tobin et al., 1990; Yang et al., 1991.)

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CAUSES OF WEANING FAILURE

✓ Weaning failure is generally related to (1) increase of airflow resistance, (2) decrease of compliance, or (3) respiratory muscle fatigue.

Aside from the pathological conditions that lead to the need for mechanical ventilation, weaning failure may occur when the work of spontaneous breathing becomes too great for the patient to sustain. Weaning failure is generally related to (1) increase of airflow resistance, (2) decrease of compliance, or (3) respiratory muscle fatigue.

Increase of Airflow Resistance

✓ ET tubes of size 8 or larger should be used to reduce the airflow resistance.

Normal subjects using an endotracheal (ET) tube have an increase of 54% to 240% in the work of breathing, depending on the size of the ET tube and ventilator flow rate (Fiastro et al., 1988). An 8-mm ET tube has a cross-sectional area of 50 mm^2, which is slightly smaller than the average cross-sectional area of the adult glottis (66 mm^2), the narrowest part of the airway (Kaplan et al., 1991). To minimize the effects of an artificial airway on airflow resistance, ET tubes of size 8 or larger should be used when it is appropriate to the patient's size. In addition, the ET tube may be cut to about an inch from the patient's lips to minimize the airflow resistance contributed by the length of the ET tube. The cut section of the ET tube should be displayed prominently so that others would not presume that the ET tube had been moved deep into the brochus.

Other strategies for decreasing airway resistance can easily be done by periodic monitoring of the ET tube for kinking or obstructions by secretions, or other devices attached to the ET tube such as a continuous suction catheter, heat and moisture exchanger, or end-tidal CO_2 monitor probe. Endotracheal suctioning to remove retained secretions and use of bronchodilators to relieve bronchospasm have also been used successfully to reduce the airflow resistance.

Decrease of Compliance

✓ Low lung or thoracic compliance makes lung expansion difficult, and it is a major contributing factor to respiratory muscle fatigue and weaning failure.

Abnormally low lung or thoracic compliance impairs the patient's ability to maintain efficient gas exchange. Low compliance makes lung expansion difficult and, it is a major contributing factor to respiratory muscle fatigue and weaning failure.

In situations where the compliance gradually decreases (e.g., ARDS), the resultant refractory hypoxemia and increased work of breathing may lead to muscle fatigue and ventilatory failure. When this occurs to a patient undergoing a weaning trial, a return to the mechanical ventilator is almost inevitable. Table 16-8 shows some examples that lead to a decreased compliance measurement.

Respiratory Muscle Fatigue

Respiratory work is a product of transpulmonary pressure (P_{TP}) and tidal volume (V_T). Studies have been done to evaluate the relationship between the work of breathing and a patient's ability to sustain adequate spontaneous ventilation.

$$\text{Work of breathing} = P_{TP} \times V_T$$

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TABLE 16-8 Clinical Conditions That Decrease the Compliance

Type of Compliance	Clinical Conditions
↓ Static compliance	Atelectasis ARDS Tension pneumothorax Obesity Retained secretions in lungs
↓ Dynamic compliance	Bronchospasm Kinking of ET tube Airway obstruction Retained secretions in airways

The transpulmonary pressure is increased in conditions of low compliance or high airway resistance. Normally a threshold work value of 1.6 kg.m/min or less is needed before ventilator-dependent patients can be weaned and assume adequate spontaneous breathing. Conditions leading to an increased workload such as low compliance and high airflow resistance may lead to respiratory muscle fatigue and eventual ventilatory failure. A threshold work value of 1.7 kg.m/min or higher is associated with failure to wean from mechancial ventilation (Tobin et al., 1990; Vassilakopoulos et al., 1996).

Prolonged full ventilatory support and muscle disuse may lead to respiratory muscle dysfunction and diaphragmatic atrophy. The cellular mechanism for the rapid onset of mechanical ventilation-induced (MV-induced) diaphragmatic atrophy is unclear. Studies have shown MV-induced oxidative stress is an important contributor to MV-induced proteolysis and contractile dysfunction (Betters et al., 2004; Levine et al., 2008). Other factors that may contribute to muscle weakness include inadequate oxygen delivery (low O_2 content or cardiac output), insufficient nutrition or electrolyte imbalance, especially hypokalemia, hypophosphatemia, hypocalcemia, and hypomagnesemia (Knochel, 1982).

Retraining of atrophied muscles may be accomplished by short T-tube trials that improve respiratory muscle strength. Pressure support ventilation may also be tried as it increases diaphragmatic endurance (Hess et al., 1991).

TERMINAL WEANING

Terminal weaning is defined as withdrawal of mechanical ventilation that results in the death of a patient. This differs from withholding of mechanical ventilation in which the patient is not placed on any mechanical ventilatory support.

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Terminal weaning is defined as withdrawal of mechanical ventilation that results in the death of a patient.

Decisions to withdraw life-support measures (e.g., mechanical ventilation, nutritional support) have become more common. This trend is partly due to the public's awareness of the quality-of-life issue, and their knowledge that death is an inevitable process in spite of medical advances, state-of-the-art medical equipment, and pulmonary rehabilitation strategies (Jacavone & Young, 1998). It is also partly due to the availability of living wills, advanced directives, and other options available to the patient and family members.

When terminal weaning is considered, four concerns must be evaluated and discussed, where appropriate, with the patient and family members: (1) patient's informed request, (2) medical futility, (3) reduction of pain and suffering, and (4) fear and distress (Campbell et al., 1992; Campbell, 2007).

Discussions on a patient's informed consent should be done over a period of time so that emotion, pain, and other intangible factors do not interfere with an informed and valid decision.

A patient's informed consent means that patients agree to have the life-sustaining devices removed, and that they understand the potential consequences (including death). No matter who initiates the discussion, the talk with patients must be open and honest. These discussions should also be done over a period of time so that emotion, pain, and other intangible factors do not interfere with an informed and valid decision.

Terminal weaning may be justified if medical intervention is futile or hopeless.

Terminal weaning may be justified if medical intervention is futile or hopeless. The interpretation of futility (hopelessness) is based on the past experience of the primary physician or specialist. Schneiderman et al. (1990) suggested that medical treatments may be futile if physicians have concluded that in the last 100 similar cases the treatments were useless. This type of objective assessment may be helpful to the patients or family members who have reservations about terminal weaning and uncertainties about the chances of recovery.

Another reason for terminal weaning is to stop pain and suffering.

Another reason for terminal weaning is to stop pain and suffering associated with the disease process (e.g., cancer), medical treatments (e.g., radiation therapy), medical procedures (e.g., arterial puncture), and psychological trauma (e.g., being totally dependent on others in an unfamiliar surrounding, unable to care for oneself, to eat, or to talk).

Physical or verbal clues may uncover that the patient is experiencing distress or fear of dying. Premedication or medication during and after withdrawal of mechanical ventilation with analgesics and sedatives can be useful in this situation. Dosage of these medications should be titrated to meet the patient's need.

Terminal weaning carries many compassionate considerations as well as ethical and legal implications (Brody et al., 1997; Campbell et al., 1992). Each health care facility should have resource persons and a standard protocol on terminal weaning available to the patients and family members, preferably before the needs arise. It is beyond the scope of this section to cover the ethical implications of terminal weaning in detail. The readers are encouraged to seek other medical ethics resources to learn more about this topic.

Prior to Withdrawal

Prior to withdrawal of mechanical ventilation, all immediate caregivers who are uncomfortable with the process should be offered an opportunity to withdraw from the case. The patient's pastor or hospital-based chaplain should be notified. The room should provide a quiet atmosphere and unlimited visitations. All invasive monitoring devices/alarms and unnecessary lines and tubes should be removed. The equipment and supplies that remain should include only the basic vital-sign monitoring devices, oxygen therapy, and intravenous access for administration of analgesics and sedatives.

Withdrawal

During the withdrawal process, the family members should be offered an opportunity to stay with the patient. The attending physician and chaplain are encouraged to be present. Analgesics and sedatives should be provided in sufficient quantity for patient comfort and relief of anxiety. The ventilator settings may be adjusted to provide minimal support (e.g., oxygen and humidity only) while the patient is still intubated. If the ventilator is turned off at this point, the patient is extubated and put on an oxygen mask to minimize prolonged hypoxia (Seton Healthcare Network, 2005). The airway is suctioned to ease patient's breathing efforts. It is important to document the events following completion of terminal weaning, per physician's pronouncement.

SUMMARY

Weaning from mechanical ventilation is not always easy because there are no absolute criteria that can guarantee successful weaning every time. The criteria for weaning provided in this chapter are based on results of research studies and experience of clinical trials from many sources. While these criteria cannot be expected to be accurate at all times, they are nevertheless very useful as a guide and a starting point for weaning trials.

From the review of available literature, it is reasonable to conclude that the more weaning criteria that are met by a patient, the more likely the weaning process will be successful. In addition to using as many clinical parameters as feasible, the patient's progress should also be monitored on a continuing basis. From these data and trends, changes and adjustments on the ventilator and treatment plan may then be made to enhance the weaning outcome.

Self-Assessment Questions

1. Which of the following patient conditions is the *least important* consideration prior to weaning a patient from mechanical ventilation?

A. frequent arrhythmias
B. ventilatory failure
C. severe acidosis
D. use of positive end-expiratory pressure (PEEP)

2. The physician asks a therapist to evaluate the following ventilatory parameters for a possible weaning attempt. The therapist should report to the physician that the only parameter that suggests successful weaning is:

   A. spontaneous frequency (f) = 40/min.
   B. spontaneous V_T = 7 mL/kg.
   C. minute ventilation = 16 L.
   D. $PaCO_2$ = 55 mm Hg.

3. The oxygenation status of a 30-year-old postoperative patient shows: PaO_2 = 55 mm Hg, SaO_2 = 85%, Q_S/Q_T = 25%, PaO_2/F_IO_2 = 118 mm Hg, F_IO_2 = 55%. Based on these data, the therapist should recommend to the physician that weaning _______ begin because _______ of the measurements presented above are within the recommended thresholds for a weaning attempt.

   A. should, 50%
   B. should, all
   C. should not, none
   D. should not, 50%

4. All of the following pulmonary measurements suggest readiness for weaning attempt *except*:

   A. maximal inspiratory pressure = −12 cm H_2O.
   B. V_D/V_T = 40%.
   C. static compliance = 32 mL/cm H_2O.
   D. f/V_T = 80 breaths/min/L.

5. A mechanically ventilated patient has the following oxygen content measurements: CcO_2 = 21.1 vol%, CaO_2 = 18.8 vol %, CvO_2 = 14.4 vol %. The calculated shunt is about _______ and it is interpreted as _______ shunt.

   A. 17%, mild
   B. 17%, significant
   C. 34%, severe
   D. 34%, significant

6. The PaO_2 of a mechanically ventilated patient is 250 mm Hg on 100% oxygen. If the P_AO_2 is 680 mm Hg, what is the alveolar-arterial oxygen tension gradient? What is the estimated shunt if every 50 mm Hg difference approximates 2% shunt?

   A. 430 mm Hg, 8%
   B. 430 mm Hg, 17%
   C. 940 mm Hg, 17%
   D. 940 mm Hg, 34%

7. Ms. Warren, a mechanically ventilated patient recovering from drug overdose, has a PaO_2 of 76 mm Hg on 30% oxygen. What is the PaO_2/F_IO_2 (P/F) index? Is the P/F index normal based on the oxygenation criteria for weaning?

   A. 25.3, normal
   B. 25.3, abnormal
   C. 253, normal
   D. 253, abnormal

8. A patient has the following measurements while receiving mechanical ventilation: corrected tidal volume = 480 mL, peak inspiratory pressure = 45 cm H_2O, plateau pressure = 30 cm H_2O, PEEP = 8 cm H_2O. What is the calculated static compliance? Is it normal based on the pulmonary measurement criteria for weaning?

   A. 11 mL/cm H_2O, normal
   B. 11 mL/cm H_2O, abnormal
   C. 22 mL/cm H_2O, normal
   D. 22 mL/cm H_2O, abnormal

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9. Rapid shallow breathing causes a(n):

A. increase of Q_S/Q_T.
B. decrease of Q_S/Q_T.
C. increase of V_D/V_T.
D. decrease of V_D/V_T.

10. A patient has an average spontaneous frequency of 20/min and tidal volume of 400 mL. What is the calculated RSBI (f/V_T)?

A. 50 breaths/min/L
B. 80 breaths/min/L
C. 200 breaths/min/L
D. 800 breaths/min/L

11. For a successful weaning outcome, a spontaneous breathing patient should have a calculated RSBI (f/V_T) of:

A. >100 breaths/min/L.
B. <100 breaths/min/L.
C. between 100 and 200 breaths/min/L.
D. between 200 and 400 breaths/min/L.

12. Partial ventilator support via _______ is done by reducing the ventilator frequency gradually.

A. synchronized intermittent mandatory ventilation (SIMV)
B. T-tube
C. spontaneous breathing trial
D. pressure support ventilation (PSV)

13. According to the weaning protocol for mechanical ventilation, the time limit for a spontaneous breathing trial should be up to _______ unless terminated earlier.

A. 5 min
B. 30 min
C. 2 hours
D. 4 hours

14. All of the following trends may be used as indicators of weaning failure with the *exception* of:

A. increasing V_T.
B. increasing $PaCO_2$.
C. decreasing PaO_2.
D. decreasing static compliance.

15. Terminal weaning is defined as _______ of mechanical ventilation that results in the _______ of a patient.

A. withholding, vegetative state
B. withholding, death
C. withdrawal, vegetative state
D. withdrawal, death

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Answers to Self-Assessment Questions

1. D.
2. B.
3. C.
4. A.
5. C.
6. B.
7. C.
8. D.
9. C.
10. A
11. B.
12. A.
13. B.
14. A.
15. D.

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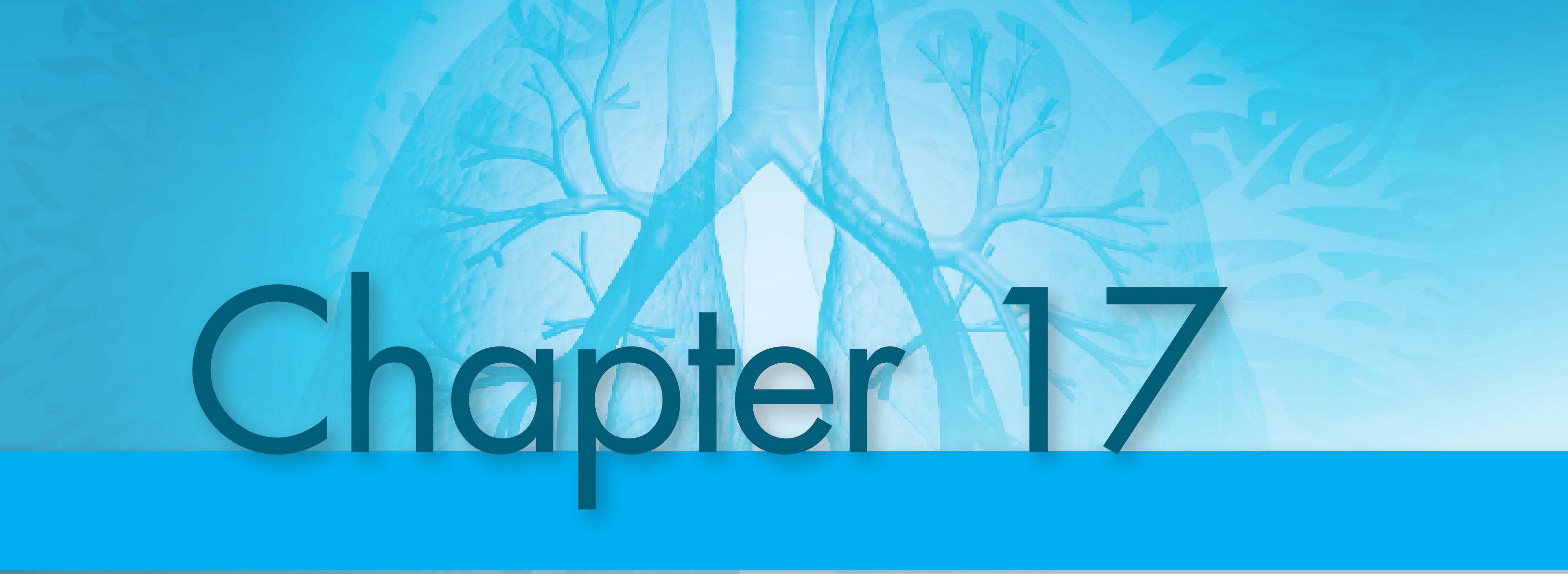

Chapter 17

Neonatal Mechanical Ventilation

Kent B. Whitaker
Lisa M. Trujillo

Outline

- Introduction
- Intubation
  - *Indications*
  - *Equipment*
- Surfactant Replacement Therapy
  - *History*
  - *Indications*
  - *Types of Surfactant and Dosages*
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- Basic Principles of Neonatal Ventilation
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  - *ECMO Criteria*
  - *Mechanisms of Bypass*
  - *Complications*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

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Key Terms

compression factor
extracorporeal membrane oxygenation (ECMO)
high frequency jet ventilation (HFJV)
high frequency oscillatory ventilation (HFOV or HFO)
high frequency ventilation (HFV)
mean airway pressure (mPaw)
nasal CPAP (N-CPAP)
power
pressure-controlled ventilation
surfactant
surfactant replacement
volume-controlled ventilation

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- List the indications for neonatal intubation and surfactant replacement therapy.
- List the types, generic names, trade names, and dosages of artificial surfactants.
- Describe the clinical application of CPAP.
- Differentiate the factors affecting the use of pressure-controlled ventilation and volume-controlled ventilation.
- Discuss the indications and initial settings for neonatal mechanical ventilation.
- Differentiate between HFPPV, HFJV, and HFOV.
- Outline the initial HFOV settings and the changes of ventilator settings based on patient condition.
- Discuss the indications, patient selection, and clinical application of ECMO.

INTRODUCTION

Neonatal mechanical ventilation provides a unique challenge to many respiratory care practitioners because neonates have different physiologic needs and laboratory values from those of adults. Also, caring for neonates often requires very different respiratory care equipment, supplies, and techniques. Because neonates are unable to communicate, keen senses and the ability to observe and evaluate neonates in distress is vital to providing appropriate care.

This chapter provides a working outline of respiratory care procedures in caring for a neonate in distress. These procedures focus on neonatal care and range from intubation and surfactant instillation immediately after birth to different types of neonatal mechanical ventilation.

INTUBATION

In most instances, intubation of the trachea is a necessary part of mechanical ventilation of the neonate. Although this chapter will discuss basics of intubation, it is beyond the scope of this chapter to cover the procedure in detail. Those interested in studying the procedure are referred to any of the several excellent neonatal/pediatric respiratory texts for that information. Here, the indications, equipment, and general considerations of neonatal intubation are covered.

Indications

✓ Intubation is indicated in (1) inadequate or prolonged bag-mask ventilation; (2) low heart rate during chest compressions; (3) endotracheal administration of epinephrine or surfactant; (4) the presence of meconium at delivery in a nonvigorous neonate (slow heart rate (<100/min), inadequate respirations, and/or poor muscle tone); and (5) extreme prematurity.

Endotracheal intubation of a neonate is indicated when (1) prolonged or inadequate bag-mask ventilation is present; (2) chest compressions are needed and heart rate remains low; (3) endotracheal administration of epinephrine or surfactant is indicated; (4) meconium is present at delivery and the neonate is nonvigorous, meaning the neonate has a slow heart rate (<100/min), inadequate respirations, and/or poor muscle tone. Intubation needs to be done as quickly as possible to remove meconium from the airway before manual ventilation; and (5) the neonate is extremely premature and mechanical ventilation is inevitable.

Other less common indications may include presence of obstructive lesions such as trachealmalacia, tracheal web, tracheal stenosis, laryngeal paralysis, and extrinsic masses. Diaphragmatic hernia, removal of pulmonary secretions, maintaining the airway during surgery, and obtaining tracheal aspirates also require intubation.

Table 17-1 shows the method of assessing a neonate using the Apgar score. Parameters listed are assigned points based on findings shown in the table. Resuscitation of a neonate should continue while scoring the Apgar. The scores range from 0 to 10 and they are given at 1 and 5 min. Scoring stops if the score is 7 or higher at 5 min. However, scoring should continue every 5 min up to 20 min if the score is less than 7.

TABLE 17-1 Apgar Score

	0	**1**	**2**
Heart rate	None	Slow (<100) Irregular	Over 100
Respiratory effort	Apnea	Irregular, shallow, gasping	Good, crying
Muscle tone	Flaccid	Some flexion of extremities	Well flexed
Reflex	No response to stimulus	Grimace	Cough or sneeze
Color	Pale blue	Blue extremities, pink body	Pink all over

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Equipment

✓ Use laryngoscope blade size 1 for most term newborns, size 0 for preemies and size 00 for micropreemies.

The basic equipment and supplies for intubation include a laryngoscope, an appropriately sized laryngoscope blade with light, and an endotracheal tube (ETT). Miller blade sizes of 1, 0, and 00 are used to intubate most term newborns, preemies and micropreemies, respectively. Selection of an ETT is based on the birth weight or gestational age of the neonate. Table 17-2 shows the guidelines for selecting an ETT for neonates as published in the Neonatal Resuscitation Program (NRP) by the American Academy of Pediatrics and American Heart Association (2006). These guidelines are commonly used by hospitals with labor and delivery rooms.

Other essential equipment may include airway suctioning devices, adhesive tape or other ETT stabilizers, flow inflating bag with 200 to 450 mL capacity and a flow restricting valve, airway manometer, and masks of different sizes (i.e., preemie, neonate, and infant) to fit over the tip of chin, mouth, and nose. The use of a T-piece resuscitator is a reliable option, in lieu of a bag and mask resuscitator, to provide ventilation to a neonate. An oxygen blender should be able to deliver a wide range of F_IO_2 (up to 100% oxygen), so weaning from oxygen may be guided by SpO_2 readings and clinical signs. The SpO_2 for premature infants less than 32 weeks gestation should be maintained between 85% and 92% to minimize the incidence of retinopathy of prematurity (ROP).

✓ Each intubation attempt should be limited to 20 sec. Bag-mask ventilation with oxygen must be done between attempts.

Intubation. Intubation should be done by two persons. The person performing the intubation should be responsible for bag-mask ventilation, intubation, and stabilization of the ETT. Another person may help by passing the intubation equipment, monitoring the patient and vital signs, and timing of the procedure. Each intubation attempt should be limited to 20 sec, and bag-mask ventilation with oxygen between attempts must be done to maintain acceptable SpO_2 and vital signs.

The infant should be put in a sniffing position and delivered free-flow oxygen during the procedure. The blade should sweep the tongue from right to left as the blade is placed into the infant's mouth. Gently advance the blade until the tip lies just beyond the base of the tongue. Lift the blade slightly by raising the entire blade, not just the tip. Look for landmarks. The vocal cords should appear as vertical stripes on each side of the glottis. Suction if necessary. Apply cricoid pressure as needed

TABLE 17-2 Selection of Neonatal Endotracheal Tubes

Tube Size (ID mm)	Weight	Gestational Age
2.5	Below 1,000 g	Below 28 weeks
3.0	1,000 to 2,000 g	28 to 34 weeks
3.5	2,000 to 3,000 g	34 to 38 weeks
3.5–4.0	Above 3,000 g	Above 38 weeks

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✓ The vocal cord marking on the ETT should stop at or slightly beyond the vocal cords.

✓ A rule to estimate the depth of intubation is to add 6 to the body weight in kilograms.

to better visualize the vocal cords and advance the ETT through the vocal cords under direct vision. If the cords are closed, wait for them to open before attempting to advance the ETT. Hold the tube against the baby's palate while removing the laryngoscope blade and stylette (if used) and securing the ETT (AAP/NRP, 2005).

If the tube has a vocal cord marking, it should be at or slightly beyond the vocal cords. If the tube does not have a vocal cord marking, a rule to estimate the depth of intubation is to add 6 to the body weight in kilograms (kg). For example, the depth of ETT for a 2-kg infant should be 8 cm (6 + 2) at the lips.

Listen for breath sounds over both lung fields and place a CO_2 detector on the ETT and verify change in color. However, a CO_2 detector should not be used when intubating for meconium removal. After confirming endotracheal placement, the tube is taped securely. A chest radiograph is done to confirm the depth of ETT placement.

SURFACTANT REPLACEMENT THERAPY

✓ The primary cause of RDS is surfactant deficiency.

surfactant: A natural phospholipid that lowers the surface tension of the lungs. Deficiency of surfactant causes high surface tension in the lungs and increases the work of breathing.

It has long been understood that the primary dysfunction in respiratory distress syndrome (RDS) is abnormally high alveolar surface tension resulting from a lack of pulmonary **surfactant**. Thus it became an item of major interest in the scientific community to develop a surfactant that can be administered to an infant to replace that which is lacking (Robertson & Halliday, 1998).

Naturally occurring surfactant is composed of several phospholipids and lipids, and four or more specific apoproteins. Each component appears to have its own distinct characteristics with regard to production, secretion, and removal (Jobe & Ilkegami, 1993). These factors have made it difficult to produce an ideal replacement surfactant.

Approximately 90% of surfactant is phospholipid, with phosphatidylcholine (PC) comprising 85% of the total amount. Roughly 60% of the PC is dipalmitoyl phosphatidylcholine (DPPC). It is the DPPC that allows surfactant to lower the surface tension of alveoli (Holm & Waring, 1993). The remaining phospholipids are phosphatidylglycerol (PG) and phosphatidylinositol (PI). Cholesterol is the predominant neutral lipid in surfactant. The four proteins found in surfactant, given the names of surfactant proteins A, B, C, and D (SP-A, etc.), make up 5% to 10% of the total. While small in quantity, their presence is essential for proper activity of pulmonary surfactant (Holm & Waring, 1993).

History

Early studies were discouraging because researchers could not find the right combination of components that formed an effective surfactant. Effective dosages and ideal method of delivery were two other questions that hindered the development of **surfactant replacement** therapy.

surfactant replacement: Direct instillation of synthetic surfactant (Surfaxin) or natural surfactant (Survanta, Infasurf) into the trachea.

Early surfactants were made with DPPC and were nebulized into the trachea. This type of surfactant alone and *method* of delivery did not produce the desired results. Continued research and later studies of surfactant and its biochemical and biophysical properties illustrated the important role of the other proteins and lipids.

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✓ Surfactant is given by direct instillation into the trachea.

New surfactants were developed that included the additional lipids and proteins. Delivery was changed from nebulization to direct instillation of surfactant into the trachea at higher dosages than had previously been used. These discoveries had dramatic effects on the surfactant-deficient premature lung, with rapid weaning of pressures and F_IO_2 levels.

Indications

✓ Prophylactic use of surfactant is indicated for infants who are at high risk of RDS, $<$26-week gestation, $PaO_2/P_AO_2 < 0.22$, or birth weight $<$1,250 g.

There are two protocols for the administration of surfactant, prophylactic and therapeutic (“rescue”). Prophylactic use of surfactant is indicated for infants who are at high risk of developing RDS because of the short gestation and low body weight. However, routine treatment of infants at risk based on these two criteria may unnecessarily commit some infants to the complications of intubation, mechanical ventilation, and surfactant administration. At one medical institution, infants born at or less than 26 weeks gestation and with $PaO_2/P_AO_2 < 0.22$ are treated prophylactically on a routine basis (British Columbia). Birth weight of less than 1,250 g may also be an indication for prophylactic use of surfactant (Survanta drug insert). Since protocols vary greatly among institutions, infants with gestational age or birth weight outside these criteria must be monitored and evaluated for possible inclusion.

✓ Prophylactic use of surfactant is also given to micropreemies ($<$30 weeks, from 500 g to 1,000 g).

Prophylactic use of surfactant is also given to micropreemies in many neonatal intensive care units (NICUs). These infants are usually $<$30 weeks and typically weigh from 500 g to 1,000 g (1 kg). They are intubated in the delivery room within the first few minutes of birth and given surfactant through the ETT within 15 minutes of delivery once ETT placement is verified by chest X-ray (Walsh et al., 2010). Pulse oximeter, oxygen blender, and the use of Neopuff, a device that allows delivery of set PIP and PEEP, should be considered because of the rapid changes in oxygenation and compliance. In many cases, rapid weaning of F_IO_2 to 21% and minimal pressures for ventilation is possible due to the rapid improvement in compliance. Many of these premature infants are extubated and transitioned to CPAP or high-flow devices such as Vapotherm.

✓ Therapeutic (“rescue”) use of surfactant is indicated in RDS (grunting, nasal flaring, retraction , cyanosis), increasing oxygen or ventilatory requirement, and positive chest radiograph.

Infants eligible for therapeutic (rescue) use of surfactant should fulfill the clinical and radiographic criteria for a diagnosis of RDS. The clinical signs may include grunting, nasal flaring, retraction, and cyanosis along with an increasing oxygen or ventilatory requirement (i.e., from CPAP to mechanical ventilation). The bilateral ground glass appearance on the chest radiograph supports the diagnosis of RDS. Infants who exhibit these clinical and radiographic signs are usually born at $<$34 weeks gestation and with an arterial/alveolar (PaO_2/P_AO_2) ratio of <0.22 (British Columbia). The indications for prophylactic and therapeutic use of surfactant are summarized in Table 17-3.

Types of Surfactant and Dosages

Currently used surfactants fall into one of two categories: those synthetically produced or obtained and processed from mammalian lungs. Surfaxin (lucinactant) is a

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TABLE 17-3 Indications for Surfactant Administration

Application	Criteria
Prophylactic use	1. Gestational age ≤26 weeks, PaO_2/P_AO_2 <0.22, <1,250 g., or 2. Gestational age <30 weeks, PaO_2/P_AO_2 <0.22, 500 g to 1,000 g. 3. Given within first 15 min of life, before symptoms appear.
Therapeutic (rescue use)	1. Signs of RDS (grunting, nasal flaring, retraction, cyanosis) 2. Positive chest radiograph for RDS (bilateral ground glass appearance) 3. Increasing oxygen requirement PaO_2/P_AO_2 <0.22 4. Increasing ventilatory requirement (from CPAP to mechanical ventilation) 5. Gestational age generally <34 weeks

synthetic surfactant approved by the FDA in 2012. Surfaxin is supplied in glass vials containing 8.5 mL of intratracheal suspension and each mL has 30 mg of phospholipids, 4.05 mg of palmitic acid, and 0.862 mg of sinapultide (rxlist.com, 2012). The first synthetic surfactant, Exosurf (colfosceril palmitate) has been discontinued in 2008 and is no longer available. There are two groups of natural surfactants that are extracted from cows and pigs. From the cow (bovine) lung tissues include Survanta®, Infasurf®, and Alveofact®. From the pig (porcine) lung tissue is Curosurf®. In 2012, the FDA approved another synthetic surfactant for use in the U.S. Surfaxin (lucinactant) is supplied in glass vials containing 8.5 mL of intratracheal suspension with each mL containing 30 mg of phospholipids, 4.05 mg of palmitic acid, and 0.862 mg of sinapultide (rxlist.com, 2012). The first synthetic surfactant, Exosurf (colfosceril palmitate) has been discontinued in 2008 and is no longer available in the U.S. A randomized trial did not find any advantage in using synthetic or natural surfactant (Horbar et al., 1993). Both surfactants have been found to be effective for the treatment of RDS (Modanlou et al., 1997; rxlist.com, 2012).

✓ Surfaxin is a synthetic surfactant. Survanta®, Infasurf®, and Alveofact® are naturally produced from cow lung tissue. Curosurf® is produced from pig lung tissue.

The recommeded dose of Surfaxin is 5.8 mL/kg birth weight. It is administered intratracheally. Up to 4 total doses of Surfaxin may be administered during the first 48 hours of life. Any 2 doses should be given at least 6 hours apart.

The recommended dosage for prophylactic or rescue use of Survanta (beractant) is 4 mL/kg birth weight. (Each milliliter of Survanta contains 25 mg of active ingredient.) Up to 4 total doses of the same amount may be given at least 6 hours apart (Witek & Schachter, 1994; Robertson & Halliday, 1998).

Table 17-4 summarizes the recommended dosages of some commonly used surfactants. This table includes synthetic and natural surfactants produced from bovine or porcine sources.

TABLE 17-4 Recommended Dosages of Common Surfactants

Type of Surfactant	Dosage
	Synthetic
Surfaxin (lucinactant)	5.8 mL/kg (Up to 4 total doses in the first 48 hours of life; any 2 doses given at least 6 hours apart.
	Natural
(A) Bovine (Survanta®, Infasurf®, Alveofact®)	Survanta® = 4 mL/kg every 6 hours up to 4 total doses in the first 48 hours. Infasurf®: 3 mL/kg every 6 hours up to 4 doses.
(B) Porcine (Curosurf®)	Initial dose = 2.5 mL/kg. Up to 2 additional doses at 1.25 mL/kg every 12-hour. Maximum of repeat doses not to exceed 5 mL/kg combined. (Divide each dose into two halves and give these halves doses 30 sec apart.)

Outcomes

On the positive side, surfactant replacement therapy appears to reduce the severity of RDS, pulmonary interstitial emphysema (PIE), and epithelial necrosis (Pinar et al., 1994), reduces pulmonary vascular resistance (Kaapa et al., 1993), improves lung function (Yuksel et al., 1993), and has beneficial long-term effects on airway resistance (Abbasi et al., 1993). Additionally, synthetic surfactants such as the discontinued Exosurf may reduce the incidence of bronchopulmonary dysplasia (BPD) and intraventricular hemorrhage (IVH) (Long, 1993).

✓ Surfactant replacement therapy reduces the severity of RDS and the incidence of some related cardiopulmonary complications.

Another study found that surfactant replacement therapy may have an impact on cerebral perfusion, most likely due to direct pulmonary or hemodynamic changes (or a combination of both) (Hentschel et al., 2002). Surfactant replacement does not work on all patients. It is not known why some patients have a transient response and others have no response. Perhaps as knowledge and understanding continue to advance in this area, we will discover those unknown factors that prevent successful use of surfactant replacement therapy in all neonates.

Since coming of age, surfactant replacement therapy has dramatically decreased the mortality rate from RDS in neonates. It should now be considered the standard of care for those neonates with RDS who require mechanical ventilation (Ramanathan, 2006).

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NASAL CPAP

Some infants may be able to ventilate satisfactorily but unable to maintain sufficient oxygenation in spite of traditional oxygen therapy. If the hypoxemia is due to mild to moderate intrapulmonary shunting and the spontaneous breathing effort is adequate and sustainable, this type of hypoxemia may be corrected by applying airway pressure alone, without the need for mechanical ventilation. Continuous positive airway pressure (CPAP) is the method to apply airway pressure without any mechanical breaths. For the neonatal population, indications for CPAP include respiratory distress syndrome (RDS), apnea of prematurity, obstructive sleep apnea (OSA), viral bronchiolitis, aspiration pneumonia, meconium aspiration syndrome (MAS), congestive heart failure (CHF), transient tachypnea of the newborn (TTN), or post-operatively (Walsh et al., 2010).

Use of Nasal CPAP

Continuous positive airway pressure (CPAP) was introduced in 1971. It reduces V/Q mismatch by improving the functional residual capacity and reducing intrapulmonary shunting. CPAP was originally administered via an endotracheal tube. Over the years, **nasal CPAP (N-CPAP)** was developed and has become a less invasive procedure to reduce the F_IO_2 requirement and the need for mechanical ventilation. Recent publications have reported that N-CPAP is both feasible and effective in most very-low-birth-weight infants and those with acute respiratory failure. Early rescue treatment with surfactant replacement with a brief intubation has further increased the effectiveness of N-CPAP (Jeena et al., 2002; Malik et al., 2003).

nasal CPAP (N-CPAP): A nasal device for delivering continuous positive pressure to the airway without the need for intubation. The neonate must have adequate and sustainable spontaneous breathing effort to use nasal CPAP.

A bubble CPAP device creates CPAP by an underwater seal with vigorous bubbling. Infants receiving CPAP by an underwater seal were observed to have chest vibration at frequencies similar to high frequency ventilation (HFV). In one study, lower minute ventilation, respiratory frequency, and better gas exchange were observed when compared with ventilator-derived CPAP devices. It suggests that the chest vibrations produced with bubble CPAP may have contributed to these findings (Lee et al., 1998).

Unlike the CPAP provided by a ventilator, the flow-generating CPAP device uses flow to maintain CPAP and has no exhalation valve. Since this CPAP device does not have an exhalation valve, it may offer a lower resistance than the ventilator CPAP. There are different-sized prongs and masks for better fitting on the patient. Different-sized hats are also available for stabilization of the flow-generating attachment. The expiratory limbs of the circuit hang loose and the flow is driven out the tube opening to room air. These devices can be noisy, but the manufacturers have produced a baffle device to reduce the noise level.

All CPAP devices should be humidified. One humidification manufacturer recommends setting temperature to 39°C because of cooling effects distal to the humidification system.

✓ The initial N-CPAP settings for most infants range from 4 to 7 cm H_2O with flow rates from 5 to 10 L/min.

The initial N-CPAP settings for most infants range from 4 to 7 cm H_2O with flow rates from 5 to 10 L/min. For infants with RDS, the CPAP level should start at 6 cm H_2O. If the infant with RDS is having recurrent apnea, persistent respiratory acidosis (pH less than 7.20), or unsatisfactory PaO_2 on >50% of oxygen with N-CPAP, intubation and surfactant therapy should be considered.

BASIC PRINCIPLES OF NEONATAL VENTILATION

The primary mode of neonatal mechanical ventilation is pressure-controlled ventilation. The ventilator generates a sufficient flow and delivers variable tidal volumes by the preset pressure. Alternatively, ventilation can be achieved by using a preset volume (volume-controlled ventilation).

Pressure-Controlled Ventilation

pressure-controlled ventilation: A preset pressure is used to deliver tidal volumes; the delivered volumes are variable.

In **pressure-controlled ventilation**, a preset peak inspiratory pressure (PIP) is used to deliver the volume. Thus, pressure is constant and volume is variable, depending on the compliance and airflow resistance characteristics of the infant. A decreasing compliance or an increasing airflow resistance requires a higher pressure to maintain the same tidal volume. With the use of pressure-controlled ventilation, frequency would more likely be adjusted to maintain minute ventilation, since the strategy of setting a PIP is to protect the lung from excessive airway pressures. As the patient's condition improves (increased compliance or decreased airflow resistance), the pressure must be decreased to avoid excessive volume and pressure. An example of this observation is the successful therapeutic response to surfactant replacement. The lung compliance of the infant increases dramatically and rapidly soon after surfactant administration. The PIP must be reduced accordingly to avoid overexpansion of the lungs.

✓ Following surfactant replacement, the PIP must be monitored and reduced accordingly to avoid overexpansion of the lungs.

Volume-Controlled Ventilation

volume-controlled ventilation: A desired tidal volume is preset; the pressure needed to deliver the volume is variable.

Volume-controlled ventilation has gained popularity because the volume is preset using variable pressures. This strategy reduces the incidence of overexpansion of the lungs. For extremely low-birth-weight infants, some ventilators (e.g., Drager Babylog) can provide a tidal volume as low as 2 mL. This is an important feature when trying to maintain a range of 3–7 mL/kg for extremely low-birth-weight infants (e.g., as low as 500 g birth weight). Typically, an initial tidal volume of 5 mL/kg is used to ventilate these infants, and the volume may be titrated up or down to meet the infant's needs. It is important to note that although volume remains constant, pressure will vary, which may result in excess pressures being delivered to the infant as a result of a decrease in compliance or increase in airway resistance. Careful setting of peak pressure alarms will aid in preventing excessive pressures being delivered in volume-controlled ventilation.

Ventilator Circuits and Humidifiers

During mechanical ventilation, some of the ventilator volume is "lost" within the circuit and humidifier and is not delivered to the patient. This wasted volume is called the compressible volume, and it is partly caused by the positive pressure applied to these devices. Higher inspiratory pressures would cause the circuit and humidifier to expand more than those under low pressures. This compressible volume is also dependent on the characteristics of the circuit and humidifier. More compliant circuits and humidifiers would expand more under positive pressure than those with low compliance.

compression factor: The amount of expansion of the ventilator circuit or humidifier during the inspiratory phase measured in mL/cm H_2O. This volume is considered "lost" and unavailable to the patient.

Circuit Compression Factor. Neonatal ventilator circuits should have a low **compression factor**. This provides minimal circuit expansion when pressure is applied during inspiration. A highly compliant circuit would expand under pressure and hold a larger portion of the volume of gas that should be delivered to the patient.

Humidifiers. Humidifiers that are used in a neonatal circuit should possess a low compressible volume. A higher compressible volume allows more expansion to occur under pressure. A humidifier with a high compressible volume may hold more volume than the neonate's lungs and thus greatly reduce alveolar ventilation.

✓ To minimize volume loss the circuit and humidifier used in a neonatal ventilator should have a low compressible factor or volume.

The ideal humidifiers for neonatal ventilation are types that incorporate a wick-type system, as they provide excellent warming and humidifying properties and maintain a low compressible volume.

Ideally, the temperature of the gas at the trachea should be 37°C with a water content of 44 mg/L. In a standard circuit (nonheated wire circuit), when inspired gas temperature is measured at the patient connection, the humidifier must heat the gas 3 or 4 degrees above the desired temperature to overcome the loss of heat after the gas exits the humidifier. One problem that occurs in this type of ventilator circuit is condensation or "rain-out" inside the tubing.

Rain-out occurs when the warmed and humidified gas exits the humidifier and makes contact with the cooler walls of the tubing. This causes the gas temperature to decrease and condensation to occur on the tubing wall. The water accumulated in the ventilator tubing may result in increased airway resistance, a higher risk of contamination, and the potential of accidentally draining the water into the patient's lungs. A water trap placed inline with the ventilator circuit helps to prevent these hazards.

✓ Heated wire inside the inspiratory tubing reduces condensation.

Heated Wire Circuits. To counter this problem, many ventilator circuits have a heated wire inside the inspiratory tubing that runs from the humidifier to the patient connection, shown in Figure 17-1. The heated wire is attached to a servo-controller before its entry into the circuit. The temperature of the gas is measured as it exits the canister and is controlled by the humidifier. The temperature is again measured at the patient connection by the servo-controller.

The servo heats the gas flow in the inspiratory tubing by heating the wire, which then heats the inspired gas. Both the humidifier and servo work from a negative feedback mechanism. The desired temperature becomes the set point, and as the actual temperature drops below the set point, power is increased until the temperature returns to the desired level. Newer circuits heat the expiratory gas in addition to the inspiratory flow, thus minimizing condensation in the expiratory line.

One potential problem with this system is found when the distal temperature probe is placed at the patient connection inside a heated incubator that is set at a

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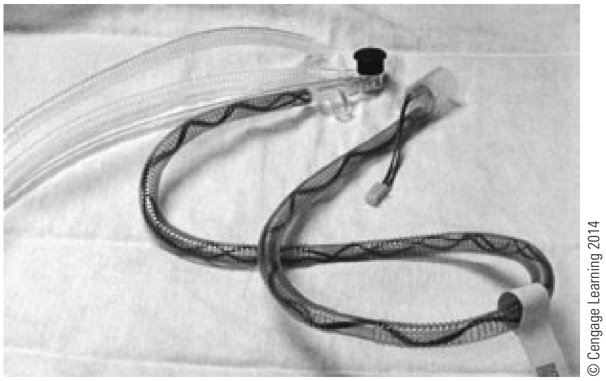
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FIGURE 17-1 Heated wire circuit inside the inspiratory tubing that runs from the humidifier to the patient connection. The heated wire is attached to a servo-controller for temperature regulation.

higher temperature than the humidifier. As the inspiratory tubing enters the incubator, the gas is heated to the set temperature of the incubator environment. This causes the temperature probe to sense the higher temperature and shut down the heater wires. The result is a buildup of condensation in the inspiratory tubing.

✓ To prevent premature shutdown (power off) of the heated wire, the temperature probe should be placed just outside the inlet to the incubator.

The solution to this problem is to place the distal temperature probe just outside the inlet to the incubator. This allows the probe to measure the actual gas temperature and properly regulate the heater wires (Chatburn, 1991).

INITIATION OF NEONATAL VENTILATORY SUPPORT

Indications for neonatal ventilatory support are based on three general guidelines: apnea, hypercapnia, and hypoxemia. These guidelines are similar to those used for adult patients. Unlike adult ventilators that use a tidal volume control to adjust the tidal volume, neonatal ventilators use a peak inspiratory pressure control to deliver an approximate tidal volume. Another unique feature of most neonatal ventilators includes the use of the flow rate and inspiratory time (I time) to fine-tune the tidal volume. The suggested initial ventilatory parameters for neonatal mechanical ventilation are discussed below.

Indications for Mechanical Ventilation

Mechanical ventilation provides two important physiologic functions. First, it maintains elastic properties and lung volumes by preventing or correcting atelectasis. By maintaining an appropriate functional residual capacity (FRC), lung compliance is maintained at an optimal level. Mechanical ventilation supplies the work of breathing when the patient is unable to maintain these properties. Second, mechanical ventilation provides the appropriate removal of CO_2 and the addition of inspired oxygen to meet the needs of the patient who cannot maintain arterial PO_2 or PCO_2 at normal levels.

✓ Mechanical ventilation is indicated when the patient is unable to maintain adequate blood gases during spontaneous breathing.

Mechanical ventilation is indicated when any condition causes a decrease in lung function and an increase in work of breathing that the patient is unable to

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TABLE 17-5 General Indications for Initiating Neonatal Mechanical Ventilation

1. Apnea a. due to prematurity b. secondary to intraventricular hemorrhage c. due to drug depression
2. $PaCO_2$ acutely rising with concurrent decrease in pH
3. PaO_2 acutely below 50 mm Hg with supplemental oxygen

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overcome. It is also indicated when the patient is unable to maintain normal blood gases during spontaneous breathing. The exact criteria used to determine the need for mechanical ventilation are difficult to define. Each institution establishes its own criteria for initiation of mechanical ventilation. Table 17-5 lists general indications for mechanical ventilation.

Initial Ventilator Settings

✔ The initial pressure setting is higher for neonates with low compliance; lower for air leaks.

The initial ventilator settings depend on several factors, including the gestational age and weight of the neonate, the disease state present, and the type of ventilator being used. In general, the smaller the neonate, the higher the incidence of barotrauma. Therefore, it is desirable to initiate these patients at lower pressures when possible. Disease states in which lung compliance is reduced will require higher initial pressures than normal or high compliance states. The presence of air leak requires low pressure and high frequency. Finally, some ventilators may require a tidal volume to be set by the clinician.

Initial Settings Based on Compliance. With respect to the above factors, Table 17-6 lists suggested starting ventilation based on compliance.

Since the tidal volume (V_T) control is not available when using pressure-controlled ventilation, an estimated V_T can be calculated by multiplying the inspiratory time (I time) and flow rate.

Example: During pressure-controlled ventilation, the I time and flow rate are set at 0.5 sec and 6 L/m, respectively. What is the calculated V_T?

$$\begin{aligned} V_T &= \text{I Time (second)} \times \text{Flow Rate (liters per minute)} \\ &= 0.5 \text{ sec} \times 6 \text{ L/min} \\ &= 0.5 \text{ sec} \times 6000 \text{ mL/60 sec} \\ &= 0.5 \text{ sec} \times 100 \text{ mL/sec} \\ &= 50 \text{ mL} \end{aligned}$$

Initial Settings Based on Birth Weight. In addition to the previous settings for neonatal mechanical ventilation, alternatives are possible by using the birth weight as a guide to determine the initial settings. Four primary parameters are discussed here for volume-controlled ventilation: frequency, inspiratory time, volume, and PEEP.

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TABLE 17-6 Initial Settings for Neonatal Mechanical Ventilation Based on Compliance

Parameter	Normal Compliance	Low Compliance
PIP	15 to 20 cm H_2O	20 to 30 cm H_2O
PEEP	3 to 5 cm H_2O	Up to 8 cm H_2O
V_T*	4 to 8 mL/kg	6 to 10 mL/kg
Frequency	25 to 40/min	Up to 150/min (esp. with air leak)
Flow Rate	6 to 8 L/min	6 to 8 L/min
I Time	0.3 to 0.5 sec	Change according to frequency to maintain an I:E ratio of 1:1
I:E Ratio	1:1.5 to 1:2	At least 1:1 (to prevent inverse I:E ratio)
F_IO_2	Set to keep patient pink with SpO_2 from 90% to 95%	Set to keep patient pink with SpO_2 from 90% to 95% Use with appropriate PEEP level if necessary

**On ventilators that are capable of providing a preset tidal volume (volume-controlled ventilation). (Data from Bandy et al., 1992; Goldsmith et al., 1993; Deakins et al., 2001; Walsh et al., 2010.)*

The frequency on many neonatal ventilators is controlled by adjusting the expiratory time after setting the flow and inspiratory time. Generally the frequency is set from 40 to 60/min for premature infants and 25 to 40/min for term infants (Table 17-7). The frequency is dependent on the ventilatory requirement and disease process. The inspiratory time is usually set at 0.3 to 0.5 sec, depending on gestational age, with a shorter inspiratory time when the frequency is high or in the presence of air trapping. Using the neonate's body weight as a guide, 5 mL/kg is the suggested initial tidal volume with a range of 3–7 mL/kg. The PEEP is set between 4 and 8 cm H_2O depending on the oxygenation status of the infant. A higher PEEP is used initially if the infant's F_IO_2 requirement is high, if there is evidence of severe

TABLE 17-7 Initial Settings for Neonatal Mechanical Ventilation Based on Weight

Parameter	Very Low Birth Weight	Low Birth Weight	Term
Frequency	40 to 60/min	40 to 60/min	25 to 40/min
Inspiratory time	0.3 to 0.5 sec	0.3 to 0.5 sec	0.3 to 0.5 sec
Volume	3 to 7 mL/kg starting at 5 mL/kg	3 to 7 mL/kg starting at 5 mL/kg	3 to 7 mL/kg starting at 5 mL/kg
PEEP	4 to 8 cm H_2O	4 to 8 cm H_2O	4 to 8 cm H_2O

intrapulmonary shunting, or if the set PEEP is not high enough to overcome critical opening pressure as indicated when viewing the compliance (pressure volume) loop. Table 17-7 provides the primary settings for volume-controlled ventilation using birth weight as a guide.

Once mechanical ventilation is initiated, the ventilator settings are fine-tuned until appropriate arterial blood gases are achieved. Blood gas measurement can be achieved by obtaining arterial blood from a peripheral or umbilical artery catheter (UAC), peripheral artery puncture, or capillary blood from a finger or heel stick. Under most circumstances, a $PaO_2 > 50$ mm Hg, a $PaCO_2$ 35 to 45 mm Hg, and a pH between 7.3 and 7.45 are acceptable for a UAC sample. An appropriately done capillary sample will roughly correlate with arterial PCO_2, pH, and HCO_3^-, and thus share the same acceptable values. A capillary PO_2 is usually not acceptable as a determinant of oxygenation status and requires the use of a pulse oximeter or transcutaneous PO_2 monitor. Common exceptions to these values are in the case of chronic lung disease patients such as pulmonary interstitial emphysema in which higher $PaCO_2$ are acceptable, and patients treated for pulmonary hypertension, where high PO_2, low PCO_2, and high pH are used.

✓ The normal arterial blood gases for neonates are $PO_2 > 50$ mm Hg, a $PaCO_2$ 35 to 45 mm Hg, and a pH between 7.3 and 7.45. For capillary samples, a lower PO_2 is acceptable.

HIGH FREQUENCY VENTILATION (HFV)

Since the explosion of research in neonatal medicine started many years ago, there is an ongoing search for a better method of ventilation to maintain adequate blood gas levels without inflicting damage on the premature lung. Several exciting methods have evolved to address these concerns.

The normally held understanding of ventilation is that the tidal volume must exceed the amount of physiologic deadspace for alveolar ventilation to occur. Conventional ventilation utilizes this principle by inflating the patient's lungs with a tidal volume that exceeds deadspace and inflates the alveoli. Expiration then occurs by the passive recoil of the thorax and lung.

high frequency ventilation (HFV): A type of ventilation that uses very high frequencies. It is subdivided into three categories: high frequency positive pressure ventilation (60 to 150 cycles per minute); high frequency jet ventilation (240 to 660 cycles per minute); and high frequency oscillatory ventilation (480 to 1,800 cycles per minute).

high frequency oscillatory ventilation (HFOV): Ventilation produced by a piston pump or loudspeaker, usually at a frequency between 480 and 1,800/min

High frequency ventilation (HFV) is a ventilation technique that delivers small tidal volumes at very high frequencies. Early studies involving HFV showed that adequate ventilation occurred even when tidal volumes far below deadspace were used (Carlo & Chatburn, 1988).

The major advantage of delivering small tidal volumes is that it can be done at relatively low pressures, greatly reducing the risk of barotrauma. In a recent study, **high frequency oscillatory ventilation (HFOV)** was compared with conventional ventilation for pulmonary dysfunction in preterm infants. The outcome resulted in a reduction in new pulmonary leaks in neonates on HFOV, although there was no significant difference in mortality rates (Bhuta et al., 2007). Despite the fact that HFV offers the advantage of oxygenation and ventilation at a lower risk of barotrauma, it has not been shown to be superior to conventional ventilation, and its use is often limited to those situations in which conventional ventilation has failed. It appears to be most useful in treating RDS and pneumonia (Clark, 1994). It is important to note that although studies regarding the comparison of HFV and

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TABLE 17-8 Classification of High Frequency Ventilation

Type Of High Frequency Ventilator	Frequency (Hertz)*	Frequency (Cycles Per Min)
High frequency positive pressure ventilation (HFPPV)	1 to 2.5 Hz	60 to 150
High frequency jet ventilation (HFJV)	4 to 11 Hz	240 to 660
High frequency oscillatory ventilation (HFOV or HFO)	8 to 30 Hz	480 to 1800

**1 Hertz (Hz) = 1 cycle per sec or 60 cycles per min.*

conventional ventilation are often inclusive, the results may be dependent upon when HFV is introduced as a means of ventilation. Early intervention may result in more desirable outcomes. Any conclusive outcomes will require additional research.

✓ HFV uses low pressures to deliver small tidal volumes. This reduces the risk of barotrauma.

HFV is delivered at frequencies between 60 and 1,800 cycles per minute (breaths per minute). The major types of HFV are categorized by the frequency of ventilation and the method with which the tidal volume is delivered. The three categories examined here are high frequency positive pressure ventilation, high frequency jet ventilation, and high frequency oscillation (Table 17-8).

High Frequency Positive Pressure Ventilation (HFPPV)

High frequency positive pressure ventilation (HFPPV) is simply conventional ventilatory breaths delivered at frequencies between 60 and 150 breaths per minute (1 to 2.5 Hz). The delivery of tidal volume during HFPPV appears to occur via convective air movement, in which tidal volume exceeds deadspace (Boynton, 1986). Modern neonatal ventilators can deliver HFPPV at frequencies up to 150/min.

✓ HFPPV is indicated on those patients who are hypoxemic or hypercapnic despite adequate and appropriate conventional ventilation.

Indications. HFPPV is indicated on those patients who are hypoxemic or hypercapnic despite adequate and appropriate conventional ventilation. Studies have shown a reduction in $PaCO_2$ and in F_IO_2 when HFPPV was used on these patients. These studies additionally showed a lower incidence of pneumothoraces in the neonates ventilated with HFPPV when compared to those receiving conventional ventilation (Boynton, 1986). There are also studies that have shown that patient-ventilator dyssynchrony may be eliminated at ventilatory frequencies of 100 to 120 breaths per minute (Milner & Hoskins, 1989).

Clinical Use. In the presence of severely noncompliant lungs, increases in peak inspiratory pressure may reach dangerous levels before an adequate tidal volume is achieved. In these cases, the frequency is increased to increase minute ventilation, allowing the peak pressure to remain lower. As frequencies increase (with sufficient flow), the inspiratory time is decreased to allow adequate exhalation of the tidal

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✔ HFPPV increases the risk of barotrauma and cardiac compromise.

volume. This requires that time constants be low to allow exhalation and prevent air trapping. Continuous monitoring of $PaCO_2$ and PaO_2 will help the practitioner achieve the frequency needed to attain the desired minute ventilation.

mean airway pressure (mPaw): The average airway pressure during a complete respiratory cycle. It is directly affected by the respiratory frequency, inspiratory time, peak inspiratory pressure, and positive end-expiratory pressure.

Hazards. As the frequency of positive pressure breaths increases, the **mean airway pressure (mPaw)** increases concurrently. The increasing mPaw greatly heightens the risk for barotrauma and cardiac compromise. The neonate is also at a higher risk of developing intracranial bleeding with increasing mPaw. The ability to suction the airway is diminished, as even short-term removal from the ventilator may result in severe hypoxemia and hypercapnia.

✔ See Appendix 1 for example.

$$\text{mPaw} = \left[\left(\frac{\text{f} \times \text{I time}}{60} \right) \times (\text{PIP} - \text{PEEP}) \right] + \text{PEEP}$$

mPaw: Mean airway pressure
f: respiratory frequency
I time: Inspiratory time
PIP: Peak inspiratory pressure
PEEP: Positive end-expiratory pressure

High Frequency Jet Ventilation (HFJV)

high frequency jet ventilation (HFJV): Ventilation through a specially designed endotracheal tube, generally at a frequency between 240 and 660/min (4 to 11 Hz).

High frequency jet ventilators generally operate at frequencies between 240 and 660/min (4 to 11 Hz). The high frequency jet ventilator delivers a high pressure pulse of gas to the patient's airway. This is done through a special adaptor attached to the endotracheal tube, or through a specially designed endotracheal tube that allows the pulsed gas to exit inside the endotracheal tube, depicted in Figure 17-2.

✔ The indications for using HFJV include severe pulmonary disease that is complicated by air leaks and other pulmonary problems.

Indications. The indications for using HFJV include severe pulmonary disease that is complicated by air leaks, such as pulmonary interstitial emphysema (PIE), pulmonary hypoplasia, restrictive lung disease, and persistent pulmonary hypertension (Gordin, 1989).

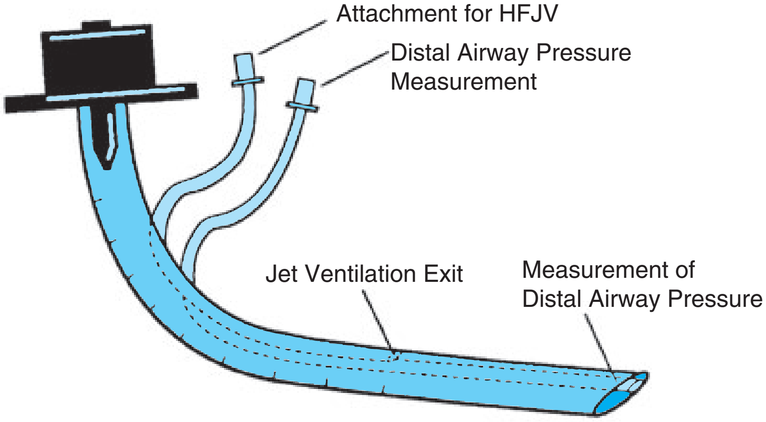
Attachment for HFJV
Distal Airway Pressure Measurement
Jet Ventilation Exit
Measurement of Distal Airway Pressure

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FIGURE 17-2 The high frequency jet ventilator (HFJV) delivers a high pressure pulse of gas to the patient's airway through a specially designed endotracheal tube that allows the pulsed gas to exit inside the endotracheal tube.

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Clinical Use. HFJV is used in tandem with conventional ventilators. The purpose of the conventional ventilator is threefold. First, it provides occasional sighs that help stimulate the production of surfactant and prevent microatelectasis. Second, the conventional ventilator provides PEEP to the patient's airway. Third, it makes a continuous flow of gas available at the endotracheal tube for entrainment by the jet ventilator (Gordin, 1989).

✓ The principal hazard of HFJV is damage to the trachea and large airways, leading to necrotizing tracheobronchitis.

Hazards. The principal hazard of HFJV is damage to the trachea and large airways, leading to necrotizing tracheobronchitis. Originally thought to be caused by poor humidification, it is now believed to be caused by the impact of the high pressure gas "bullets" on the wall of these airways (Milner & Hoskins, 1989).

To offset damage, HFJV should only be delivered through a special catheter that exits internally to the endotracheal tube or via a special triple-lumen endotracheal tube, previously mentioned. The triple-lumen tube incorporates the jet injector and a pressure monitoring port in the lumen of standard-sized endotracheal tubes.

✓ To minimize tracheal damage, the high pressure pulse of gas exits inside the endotracheal tube via a special tube.

Other hazards include gas trapping, hyperinflation, obstruction of the airway with secretions, hypotension, and inflammatory injury to the trachea (Gordin, 1989; Richardson, 1988).

✓ Transillumination of the infant chest can be used to detect tension pneumothorax.

Of concern with the use of HFJV is the difficulty encountered in assessing the patient. Auscultation of breath sounds and heart sounds is difficult due to the constant vibration and noise produced by the ventilator. Assessment of these patients is based on the observation of other clinical signs. Decreased lung compliance and pneumothoraces are observed by a decrease in chest wall vibration, increased $PaCO_2$, and a decreased PaO_2. Transillumination of the infant chest can be used to detect tension pneumothorax. A decrease in chest wall vibration and an increase in $PaCO_2$, without a drop in PaO_2, indicate an obstruction or malposition of the endotracheal tube. Microatelectasis and hyperinflation may be seen clinically as a decrease in the PaO_2. Neonates on HFJV should also be closely monitored for fluid, electrolyte, and neurological status (Gordin, 1989).

High Frequency Oscillatory Ventilation (HFOV)

High frequency oscillatory ventilation (HFOV) utilizes the highest of frequencies, usually in the range of 480–1,800/min (8 to 30 Hz). A piston pump produces the oscillatory waves that deliver the gas to the lungs.

Concept of Operation. A unique feature of HFOV is that it produces a positive as well as a negative stroke, which assists both inspiration and expiration (Figure 17-3). The HFOV device is placed inline with the endotracheal tube and a gas source is passed perpendicularly into the tube, as illustrated in Figure 17-4.

As the fresh gas enters the endotracheal tube, it is driven to the patient by the waves coming from the oscillator. Expiration occurs opposite to where the gas enters the endotracheal tube through an expiratory limb that has a high impedance to oscillations, but a low impedance when there is a steady flow of gas. Modern HFOV devices use traditional endotracheal tubes and are not used in tandem with conventional ventilators (Meredith, 1995).

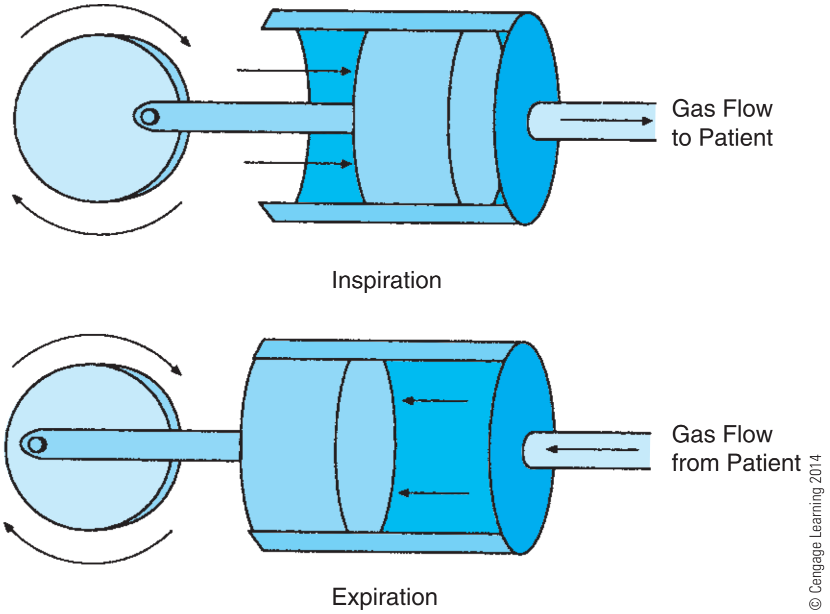
Gas Flow
to Patient
Inspiration
Gas Flow
from Patient
Expiration
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FIGURE 17-3 In high frequency oscillatory ventilation (HFOV), positive and negative strokes are provided to assist both inspiration and expiration.

Theories of Gas Exchange. A phenomenon, called pendelluft, occurs when inflated alveolar units equilibrate gases by swinging ventilations between them. This serves to improve gas exchange and impact enhanced diffusion (Higginson, 2002). With all theories of gas exchange during HFV, it may be easiest to just say "Our understanding of this complex set of gas exchange dynamics remains incomplete" (Walsh et al., 2010). Even those who experiment, compute, and mathematically examine these theories, conclude that what happens in simulations may not be what is happening in real lungs (Pedley et al., 1994). See Figure 17-4.

Indications. HFOV should be considered when conventional ventilation fails to provide adequate ventilation or oxygenation to neonates of all birth weights and gestational ages (Clark et al., 1994; Grenier & Thompson, 1996). Additionally, in recent years, studies have been done on neonates who were placed directly on HFOV and

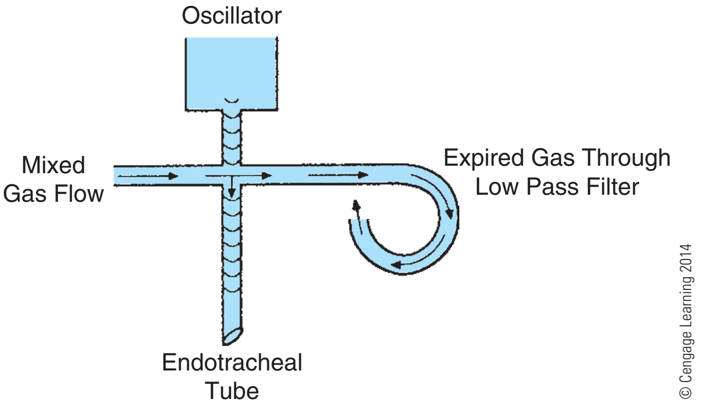
Oscillator
Mixed
Gas Flow
Expired Gas Through
Low Pass Filter
Endotracheal
Tube
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FIGURE 17-4 The oscillator or high frequency oscillatory ventilation (HFOV) device is placed in-line with the endotracheal tube and a gas source is passed perpendicularly into the tube.

intubated immediately after birth. These studies have shown to improve pulmonary outcome of very-low-birth-weight (VLBW) infants without increasing the incidence of intraventricular hemorrhage (IVH) and/or periventricular leukomalacia (PVL) (Durand et al., 2001). Since hyaline membrane disease is the most common condition in the neonatal ICU, premature infants are most likely to be considered for HFOV (Vierzig et al., 1994). The indications for HFOV are highly variable and dependent on the diagnosis and progression of the patient condition. Infants with congenital diaphragmatic hernia (Miguet et al., 1994), diffuse alveolar disease, nonhomogeneous lung disease, air leak, and pulmonary hypoplasia are potential candidates for HFOV. The major clinical conditions for HFOV are summarized in Table 17-9.

✓ Infants with congenital diaphragmatic hernia, diffuse alveolar disease, nonhomogeneous lung disease, air leak, and pulmonary hypoplasia are potential candidates for HFOV.

While HFOV can be useful to treat a variety of conditions, it is of interest to note that the presence of airleaks and lack of early improvement indicate a poor prognosis (Chan et al., 1994).

Benefits. Three benefits of HFOV have been demonstrated. First, it appears as though HFOV prevents the release of inflammatory chemical mediators in the lung, resulting in less lung injury than is seen with conventional ventilation (Imai et al., 1994). Second, when used in conjunction with surfactant replacement therapy during the first hours of life, the incidence and severity of bronchopulmonary dysplasia (BPD) may be reduced (Jackson et al., 1994). The third benefit of HFOV is that when applied early to maintain ventilation with optimal lung volume, oxygenation is increased in acute stages of RDS. This improvement in oxygenation reduces the need for surfactant administration (Plavka et al., 1999).

Complications. The ability of HFOV to oxygenate the blood is not as good as with other methods. This often requires the use of high levels of PEEP, often in excess of 15 cm H_2O (Milner & Hoskins, 1989). Combined with evidence that HFOV causes

TABLE 17-9 Clinical Conditions for HFOV

Clinical Condition	Notes
Failing conventional ventilation	Unable to maintain acceptable blood gases Deteriorating clinical condition
Increasing ventilation requirement	F_IO_2 >50%, frequency >30/min, and PIP >20 cm H_2O for infants <1,000 g (PIP in high 20 cm H_2O for infants >1,500 g)
Rapidly increasing F_IO_2 requirement (without pneumothorax)	Oxygen index >10 (e.g., patent ductus arteriosus)
Chest radiograph consistent with diffuse, homogeneous lung disease (without air trapping)	Hyaline membrane disease (HMD)
Pulmonary hypertension	Nitric oxide candidates, oxygen index ≥15

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hyperinflation of the alveoli, high levels of PEEP may compromise cardiac output and lead to a higher risk of developing barotrauma (Milner & Hoskins, 1989).

There are several technical problems encountered in the use of HFOV. One problem is in the measurement of pressure at the distal end of the endotracheal tube. It is likely that alveolar pressures are quite different from those measured at the carina. An additional problem is a general lack of HFOV devices and training for their use in level I and level II nurseries.

During HFJV and HFOV, cardiopulmonary assessment of the patient is difficult. Signs of pallor, cyanosis, bradycardia, hypotension, and increased respiratory effort indicate a worsening of status.

Initial HFOV Settings

Once an infant is committed to HFOV, the following sequence may be used to set up the ventilator: mean airway pressure (mPaw), flow, power, frequency, inspiratory time %, F_IO_2. Each of these parameters and settings is discussed below. Table 17-10 provides the initial HFOV settings. A summary of the HFOV guidelines for specific clinical conditions is provided in Table 17-11.

In addition to the F_IO_2, the mPaw affects mostly the oxygenation of the patient.

Mean Airway Pressure (mPaw). The mPaw affects mostly the oxygenation of the patient. The initial setting of mPaw on HFOV should be based on the mPaw during conventional ventilation. Generally, a higher initial mPaw is used for HMD and

TABLE 17-10 Initial HFOV Settings

Parameter	Initial Settings
mPaw (dependent on power setting)	Maintain mPaw 3 to 4 cm H_2O > during conventional ventilation for diffuse alveolar disease (e.g., HMD) Maintain mPaw ≤ less than during conventional ventilation for nonhomogeneous lung disease or air leak (permissive hypercapnia should be considered for these infants).
Bias flow	20 L/min (>2000 g) 10 to 15 L/min (<2000 g)
Power [amplitude of oscillation (ΔP) − increasing the power increase delivered volume]	Adjust power in increments of 2 to 4 cm H_2O until adequate chest wiggle
Frequency (Hz)—*decreasing* the frequency or Hz increase delivered volume	12.5 to 15 Hz (<1,000 g) 10 to 12.5 Hz (1,000 to 1,500 g) 10 Hz (>1,500 to 2,000 g) 8 to 10 Hz (>2,000 g)
Inspiratory time %	33% for an I:E ratio of 1:2
F_IO_2	Initially 100%; titrate following stabilization of the patient to maintain adequate SpO_2

(Data from Deakins et al., 2001.)

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TABLE 17-11 HFOV Guidelines for Specific Clinical Conditions*

Clinical Condition		Power (ΔP or Amplitude)	Mean Airway Pressure during Conventional Ventilation (mPaw-CV)	Frequency (Hz)
Diffuse alveolar disease	Premature infant	Adequate chest wiggle**	1 to 2 cm H_2O >mPaw-CV	10 to 15 Hz
	Term or near term	Adequate chest wiggle	2 to 4 cm H_2O >mPaw-CV	8 to 10 Hz
Nonhomogeneous lung disease	Meconium aspiration syndrome (with air trapping)	Good to adequate chest wiggle	= mPaw-CV if tolerated	6 to 10 Hz
	Meconium aspiration syndrome (diffuse)	Good to adequate chest wiggle	2 to 4 cm H_2O >mPaw-CV if tolerated	6 to 10 Hz
	Focal pneumonia	Adequate chest wiggle	= or 1 cm H_2O < mPaw-CV if tolerated	8 to 10 Hz
Air leak	Premature (PIE)	Minimal chest wiggle	1 cm H_2O <mPaw-CV if tolerated	10 to 15 Hz
	Premature (gross air leak)	Adequate chest wiggle	= or 1 cm H_2O > mPaw-CV if tolerated	10 to 15 Hz
	Term/Near Term (gross air leak)	Adequate chest wiggle	= or 2 cm H_2O > mPaw-CV if tolerated	8 to 10 Hz
Pulmonary hypoplasia	Uniform disease	Minimal chest wiggle (may need to increase to maintain PCO_2)	= mPaw-CV (may need to ↑ to improve SaO_2)	10 to 15 Hz (may need to ↓ to maintain PCO_2)
	Nonuniform disease	Minimal chest wiggle (may need to increase to maintain PCO_2)	= up to 2 cm H_2O > mPaw-CV (use good lung to guide)	10 Hz (may need to ↓ to maintain PCO_2)

These guidelines should be used as initial settings onl,y and they must be adjusted for changing clinical conditions. Appropriate patient assessment and monitoring must be provided at all times before and during HFOV.

***Chest wiggle is determined by observable chest movement down to the eight ribs or umbilicus during HFOV.*

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a lower initial mPaw is used for nonhomogenous lung disease. Changes should be made in increments of 1 to 2 cm H_2O unless the PO_2 demands require dynamic changes for increasing or decreasing the mPaw. When lung compliance and oxygenation improve, a subtle drop in the mPaw may be observed.

During weaning, changes in the mPaw should be done every 6 hours, more often if rib expansion of greater than nine ribs posterior continues.

Flow. The initial flow settings are 20 L/min for infants weighing more than 2,000 g. For infants less than 2,000 g, 10 to 15 L/min should be adequate.

power: A setting during HFOV that determines the amplitude of oscillation, tidal volume, and degree of ventilation.

✓ Changes in the power setting will affect the mPaw, thus requiring readjustment of the mPaw.

✓ The frequency may need adjustment when changes are made to amplitude or mPaw.

✓ Increasing the power (amplitude of oscillation or ΔP) or *decreasing* the frequency (Hz) increase delivered tidal volume and decrease $PaCO_2$.

Power. The **power** setting determines the amplitude of oscillation (ΔP) and thus the tidal volume and degree of ventilation. In HFOV, the tidal volume produced by the power setting is less than the deadspace volume. The CO_2 is drawn out actively during oscillation. Initially, the power setting should be increased in increments of 2 to 4 cm H_2O unless the PCO_2 demands require dynamic changes for increasing or decreasing the amplitude.

Changes in the power setting will affect the mPaw, thus requiring readjustment of the mPaw. The piston should be centered continuously when changes are made.

Frequency. The initial frequency setting is 8 to 15 Hz depending on the size of the infant and the diagnosis. The frequency may need adjustment when changes are made to amplitude or mPaw. The piston should be centered continuously when changes are made. Increasing the power (amplitude of oscillation or ΔP) or *decreasing* the frequency (Hz) increase delivered tidal volume and decrease $PaCO_2$.

Inspiratory Time %. The inspiratory time % determines the I:E ratio and is usually set at 33%. This setting provides an I:E ratio of 1:2. This parameter is not routinely changed.

F_IO_2. The initial F_IO_2 may be set at 100%. After stabilization of the patient, the F_IO_2 is titrated to keep SpO_2 between 90% and 95%.

OTHER METHODS OF VENTILATION

✓ Dual control refers to a breath type that combines the useful features of volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV).

There has been much research, development, and use of dual control ventilation. Dual control refers to a breath type that combines the useful features of volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV). Typical VCV delivers a set tidal volume and the breath type has a fixed flow rate. In high-volume demand situations, insufficient inspiratory flow may lead to patient-ventilator dysychrony. In PCV, the patient's flow requirement is supplied instantaneously. One major limitation of PCV is inconsistent tidal volume in conditions of changing airflow resistance and compliance.

Examples of dual control mode that are used in neonatal mechanical ventilation include pressure-regulated volume control (PRVC), volume-assured pressure support (VAPS), airway pressure release ventilation (APRV), machine volume (MV), and volume guarantee (VG). PRVC, VAPS, and APRV are outlined in Chapter 4. The following sections provide an overview of machine volume, volume guarantee, and liquid ventilation.

Machine Volume

Machine Volume in AVEA (CareFusion) uses the desirable features of both volume-controlled and pressure-controlled ventilation. The clinician sets a target tidal volume and inspiratory time (volume-controlled), and the ventilator will deliver the tidal volume using variable flow to meet the patient's resistance, compliance, and flow demands (pressure-controlled). The inspiratory pressure used to generate the flow is determined within the breath (while the breath is being delivered).

Machine Volume for neonatal application requires a proximal flow sensor. The initial *Machine Volume* may be set in three steps: (1) In pressure-controlled mode, adjust the peak inspiratory pressure to reach the desired tidal volume (or V_T/Kg), (2) Select V_{del} (uncorrected tidal volume during PCV) as a monitoring parameter, and (3) Set the *Machine Volume* to the V_{del} (or slightly below the V_{del} to anticipate any slight decrease in compliance).

Volume Guarantee

Volume Guarantee in Babalog 8000 plus (Dräger) can be described as pressure-limited ventilation with tidal volume targeting. It is not pressure-controlled because the inspiratory pressure for *each* breath in *Volume Guarantee* is variable (or limited), depending on the preceding *expired* tidal volume.

In *Volume Guarantee*, the clinician sets the tidal volume and maximal peak inspiratory pressure. Immediately before delivery of tidal volume breaths, the ventilator monitors the preceding expired tidal volume and makes breath-by-breath adjustments of the peak inspiratory pressure to deliver the set tidal volume. This method of breath delivery allows the lowest peak inspiratory pressure needed to deliver the set tidal volume.

Volume Guarantee is active in the PSV, SIMV, or SIPPV (synchronized intermittent positive pressure ventilation [same as assist/control]) mode. In PSV, the inspiratory time is limited by the mechanics of the baby's lungs. It is more natural because the inspiration will be terminated once the baby's lungs are full. In SIMV and SIPPV, the inspiratory time is set by the clinician. Should the set inspiratory time be longer than the baby needs to fill the lung, the inspiratory pressure is limited to the set maximum peak inspiratory pressure.

The goal of *Volume Guarantee* is to reduce volutrauma and barotrauma to the baby's lungs. The underlying premise is that using the lowest inspiratory pressure to deliver a target tidal volume will result in a lower incidence of lung injury.

Liquid Ventilation

Although not a new concept, the ability to successfully utilize this technology has been developed over the past several years. The concept behind liquid ventilation is to use liquid rather than gas to inflate the lungs, resulting in more equal distribution of ventilation. Mechanical inflation could then occur at pressures low enough to prevent damage to lung tissues.

Perfluorocarbon (PFC) liquids are the first substances that have been shown to support respiration and are able to carry more oxygen than a gas. Additionally, PFC can more easily remove carbon dioxide than a gas (Leach et al., 1996).

Liquid ventilation has potential applications for use in several diseases that traditionally have been difficult to treat. Included are RDS, aspiration syndromes, persistent pulmonary hypertension of the newborn, and pneumonia (Greenspan, 1993). While the potential of a favorable impact on the treatment of neonates is nearer, much research is still necessary before liquid ventilation takes its place among current treatment modalities.

EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO)

With patients for whom it is difficult or near impossible to maintain adequate oxygenation by conventional means (oxygen therapy, CPAP, PEEP), it may become necessary to oxygenate the blood outside the body. One method used with moderate success is the procedure of **extracorporeal membrane oxygenation** (**ECMO**).

extracorporeal membrane oxygenation (ECMO): Oxygenation of blood outside the body through a membrane oxygenator.

History

Oxygenation of blood outside the body, through a membrane oxygenator, was first developed for use in open-heart surgery in the 1950s. The technology continued to improve and modifications allowed long-term use of the technique in the 1960s (Carlo & Chatburn, 1988).

The first use of the extracorporeal membrane oxygenator on an infant was done and described in 1971 (Zwischenberger et al., 1986). This paved the way for perfection and refinement of the technique. Today ECMO is used in many institutions across the country.

Patient Selection

Because of the potential risks associated with ECMO, the clinical criteria used selects only those infants who are at an 80% or greater risk of mortality if conventional methods are used.

✓ Candidates for ECMO include patients with PPHN, MAS, sepsis or congenital diaphragmatic hernia.

Patients with severe presentations of the following pathologies may be considered as candidates for ECMO: persistent pulmonary hypertension of the newborn (PPHN), meconium aspiration syndrome (MAS), sepsis, or congenital diaphragmatic hernia.

✓ ECMO is not recommended for infants of less than 34 weeks gestational age, weighing less than 2,000 g, or having evidence of intracranial hemorrhage (ICH).

Several limitations have been established that help to define the ECMO population. Those infants at a gestational age less than 34 weeks or weighing less than 2,000 g are excluded from consideration. This is due to the significant mortality associated with intracranial hemorrhage (ICH) (Revenis et al., 1992). Any patient with an existing ICH is not a candidate for ECMO. This is because of the need for systemic heparinization during the procedure, which could worsen an intracranial bleed. This also requires that any coagulopathy be corrected before initiating ECMO.

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✓ ECMO candidates should have less than 2 weeks of ventilator assistance, and have reversible lung disease.

ECMO is contraindicated when mechanical ventilation has been used for more than 2 weeks prior to the initiation of ECMO. This is because of the likelihood of the development of chronic lung disease, which ECMO cannot reverse (Short, 1994). Additionally, candidates must have a reversible lung disease and should be free of significant cardiac disease.

ECMO Criteria

✓ The mortality rate of infants under conventional ventilator care strategies can be predicted by three methods: $P(A\text{-}a)O_2$, oxygen index, PaO_2 or pH measurements.

Since ECMO therapy is reserved for candidates with an extremely high mortality rate (80% or greater) under conventional ventilator care strategies, there are three ways to predict the occurrence of this mortality rate: (1) alveolar-arterial oxygen pressure gradient [$P(A\text{-}a)O_2$ or $A\text{-}aDO_2$], (2) oxygen index, and (3) PaO_2 or pH measurements.

Using the $P(A\text{-}a)O_2$. The $P(A\text{-}a)O_2$ is measured and calculated with the patient on 100% oxygen:

$$P(A\text{-}a)O_2 = \text{barometric pressure} - 47 - PaCO_2 - PaO_2$$

$P(A\text{-}a)O_2$ of 605 to 620 mm Hg (at 100% F_IO_2) for 4 to 12 hours indicates a need for ECMO therapy.

✓ See Appendix 1 for example.

Using the Oxygen Index. The OI is determined as follows:

$$OI = \frac{(mPaw \times F_IO_2)}{PaO_2}$$

Patients with an oxygen index of 0.35 to 0.6 for 30 minutes to 6 hours are inclusive for ECMO therapy.

A study by Durand et al. (1990) described the use of the OI in addition to mPaw to identify a select group of meconium aspiration patients for ECMO. Their conclusions were that an OI greater than 0.4 and a mPaw of 20 cm H_2O or greater, may be helpful in identifying patients who could benefit from ECMO.

Using the PaO_2 or pH. A third criterion used is the presence of a PaO_2 of 35 to 50 mm Hg for 2 to 12 hours or a pH of <7.25 for 2 hours with hypotension (Short, 1994). Table 17-12 summarizes the parameters and critical values that may be used to determine the need for ECMO therapy.

TABLE 17-12 Criteria for ECMO Therapy

Parameter	Critical Value during Mechanical Ventilation
Alveolar-arterial oxygen tension gradient ($A\text{-}aDO_2$) on 100% F_IO_2	605 to 620 mm Hg for 4 to 12 hours
Oxygen index (OI)	0.35 to 0.6 for 30 min to 6 hours
PaO_2	35 to 50 mm Hg for 2 to 12 hours
pH	<7.25 for 2 hours with hypotension

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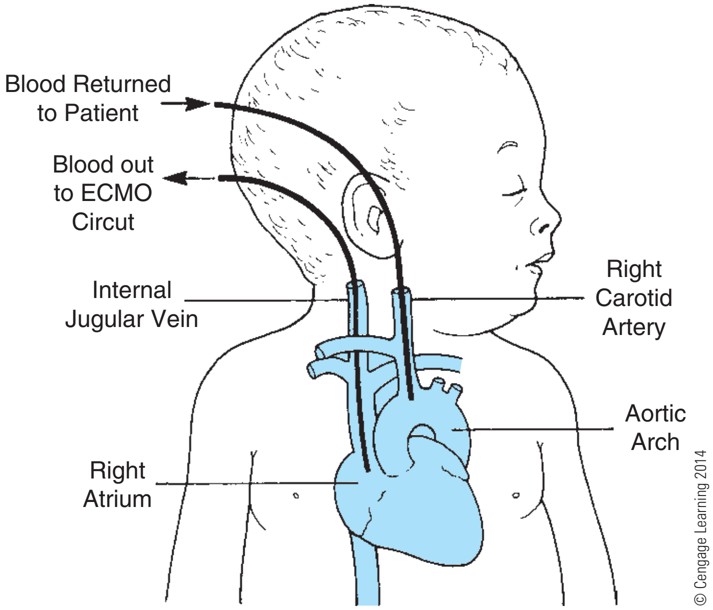

FIGURE 17-5 Venoarterial route of ECMO setup. Blood is drawn from the right atrium via the internal jugular vein. The oxygenated blood is returned to the aortic arch via the right carotid artery.

Mechanisms of Bypass

✔ In the venoarterial route, blood goes from the right atrium (via the internal jugular vein) to the aortic arch (via the right common carotid artery). This route oxygenates the blood and supports the patient's cardiac function.

✔ In the venovenous route, blood goes from the right atrium (via the right internal jugular vein) and returns to the right atrium (via the femoral vein). This route oxygenates the blood only and does not support the patient's cardiac function.

Two types of ECMO can be done: venoarterial and venovenous.

Venoarterial. In the venoarterial route, blood is drawn from the right atrium via the internal jugular vein. The oxygenated blood is returned to the aortic arch via the right common carotid artery, as shown in Figure 17-5. Venoarterial ECMO not only oxygenates the blood but also supports the cardiac function of the patient, because the blood return to the aortic arch is supported by the ECMO machine. For this reason, the venoarterial route is most commonly used for the ECMO procedure (Donn, 1990).

Venovenous. In the venovenous route, blood is removed from the right atrium via a catheter inserted in the right internal jugular vein. The oxygenated blood is returned to the right atrium through a catheter inserted via the femoral vein. This method oxygenates the blood and does not support cardiac output.

In the venovenous method, blood flow from right to left heart remains the sole function of the heart.

ECMO Circuit. The ECMO circuit uses a modified heart-lung bypass machine consisting of a venous-blood drainage reservoir, a blood pump, the membrane oxygenator where the exchange of O_2 and CO_2 takes place, and a heat exchanger to maintain temperature. Figure 17-6 depicts a typical ECMO circuit.

Complications

Complications of ECMO are both mechanical and physiologic. Common physiologic complications of ECMO are those related to bleeding, secondary to the high

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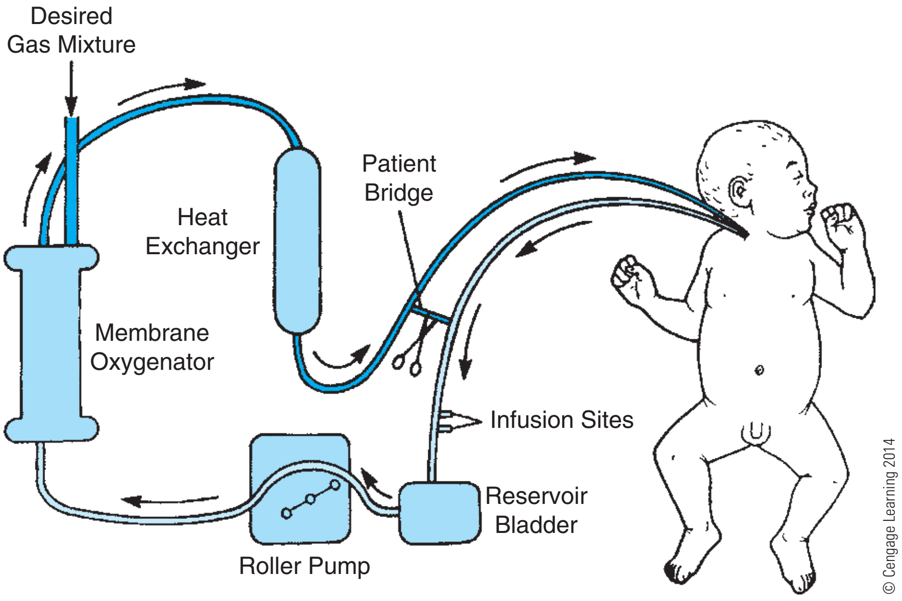

FIGURE 17-6 A typical ECMO circuit.

✔ Bleeding, ICH, pulmonary edema, and hemorrhage are some potential complications of ECMO therapy.

level of heparin required for anticoagulation. Intracranial hemorrhage (ICH) has been reported to affect 14% of ECMO patients in one study (Donn, 1990). The incidence of ICH may be decreased if cephalic jugular venous drainage is used in conjunction with ECMO (O'Connor, 1993).

There is also a high incidence of seizures in ECMO patients. It is unknown whether the seizures are caused by the therapy, the disease, or both (Donn, 1990). Pulmonary edema, the release of vasoactive substances secondary to platelet-membrane interaction, and pulmonary hemorrhage are potential pulmonary complications.

Cardiovascular complications arise from hypo- and hypervolemia leading to hypo- and hypertension in the infant. Hypertension is seen in about 7% of ECMO patients (Donn, 1990). Alteration of the reninangiotensin-aldosterone cycle, secondary to the nonpulsatile perfusion, may lead to renal complications.

Anemia, leukopenia, and thrombocytopenia are all possible hematologic complications caused by the consumption of blood components by the membrane oxygenator (Carlo & Chatburn, 1988). Due to the invasive nature of ECMO, there is an increased risk of infection. Roughly 6% of patients on ECMO have positive blood cultures (Donn, 1990).

Mechanical complications arise in approximately 10% of ECMO cases and include failure of the pump, rupture of the tubing, failure of the membrane, and difficulties with the cannulas (Donn, 1990).

Implications. The initial hope that ECMO could provide a safe means of ventilating the sick neonate has not been realized. It became apparent early on that there are many hazards and complications that make the procedure suitable in only a few selected clinical conditions. The long-term complications of ECMO are still unknown. It is necessary to permanently ligate the carotid artery used to cannulate the patient, and the effects of having a single carotid artery as the patient grows older have yet to be determined.

The ECMO procedure is expensive and requires around-the-clock monitoring by specially trained personnel, which increases the cost tremendously. The high cost is often offset, however, by a decreased number of days spent in the hospital. Patients receiving ECMO averaged 25 days in the hospital, compared to 76 days for those treated conventionally (Wagner, 1989).

SUMMARY

Neonatal mechanical ventilation is an area that requires additional training and clinical experience beyond the basics of respiratory care. Neonates should not be viewed as small adults because the ventilator settings, normal values, and treatment plans are all unique and sometimes wearisome to manage. This is particularly true for respiratory care practitioners who "wear more than one hat" and work in both the adult and neonatal intensive care units.

Since the materials presented in this chapter are relevant to actual clinical practice, they should be very useful to respiratory care students as they prepare to train and practice in the neonatal intensive care units. For seasoned practitioners, the reference sources should provide additional information on the topics presented in this chapter.

Self-Assessment Questions

1. Intubation of neonates following delivery is indicated under all of the following conditions *except*:

   A. meconium staining of amniotic fluid.
   B. difficulty ventilating by bag and mask.
   C. Apgar score greater than 8.
   D. presence of diaphragmatic hernia.

2. For neonates below 1,000 g body weight, the proper size of laryngoscope blade should be size _______ and endotracheal tube size _______ (internal diameter, mm):

   A. 0, 1.5
   B. 0, 2.5
   C. 1, 1.5
   D. 1, 2.5

3. The most common cause of respiratory distress syndrome in newborns is:

   A. surfactant deficiency.
   B. oxygen toxicity.
   C. congenital heart disease.
   D. low body weight.

4. A preterm infant has a diagnosis of respiratory distress syndrome. The therapist should expect to read in the chart that the neonate showed all of the following signs *except*:

   A. expiratory grunting.
   B. nasal flaring.
   C. apnea.
   D. chest retraction.

5. Which of the following statements is *true* regarding surfactant replacement therapy?
   - A. Survanta® is a synthetic preparation.
   - B. Exosurf Neonatal® currently available in the U.S.
   - C. Surfactant replacement works on all infants.
   - D. Surfactant replacement reduces the severity of RDS.

6. During _______ controlled ventilation, the ventilator delivers a variable _______ depending on a patient's lung compliance or airflow resistance.
   - A. pressure-, volume
   - B. pressure-, flow
   - C. volume-, flow
   - D. volume-, volume

7. During pressure-controlled ventilation, a lower tidal volume would result when the patient's compliance is _______ or airflow resistance is _______.
   - A. increased, increased
   - B. increased, decreased
   - C. decreased, increased
   - D. decreased, decreased

8. A blood gas report done on a neonate shows a $PaCO_2$ of 58 mm Hg. The physician asks the therapist to increase the delivered volume via pressure-controlled ventilation. The therapist should increase the:
   - A. tidal volume.
   - B. peak inspiratory pressure.
   - C. expiratory time.
   - D. positive end-expiratory pressure.

9. During mechanical ventilation, the volume loss in the circuit and humidifier may be minimized by using a circuit and humidifier with:
   - A. large mechanical deadspace volume.
   - B. heated wire.
   - C. low compression factor.
   - D. high compression factor.

10. A heated wire is sometimes placed on the inspiratory side of the circuit to reduce _______ in the ventilator circuit.
    - A. airway resistance
    - B. condensation
    - C. contamination
    - D. circuit temperature

11. The general indications for mechanical ventilation include all of the following *except*:
    - A. acute alveolar hyperventilation.
    - B. apnea.
    - C. acute respiratory acidosis.
    - D. acute hypoxemia (PaO_2 <50 mm Hg with supplemental oxygen).

12. When using pressure-controlled ventilation, the tidal volume delivered by the ventilator may be estimated by:
    - A. I time + Flow rate.
    - B. I time × Flow rate.
    - C. I time + E time.
    - D. Flow rate/I time.

13. The blood gas values of a normal umbilical artery sample include all of the following *except*:

A. pH from 7.3 to 7.45.
B. PCO_2 from 35 to 45 mm Hg.
C. PO_2 greater than 50 mm Hg.
D. SaO_2 from 60% to 90%.

14. High frequency ventilation has the advantages of delivering _______ and reducing the incidence of _______.

A. small tidal volume, barotrauma
B. large tidal volume, barotrauma
C. large mean airway pressure, necrotizing tracheobronchitis
D. low peak inspiratory pressure, air trapping

15 to 17. Match the type of high frequency ventilator with the respective frequency (cycles/minute). Use each answer once.

Type Of High Frequency Ventilator	Frequency (Cycles Per Minute)
15. High frequency jet ventilation (HFJV)	A. 60 to 150
16. High frequency oscillatory ventilation (HFOV or HFO)	B. 240 to 660
17. High frequency positive pressure ventilation (HFPPV)	C. 480 to 1800

18. To minimize tracheal damage caused by high pressure gas jets in high frequency jet ventilation (HFJV), a specially designed tube allows pulsed gas to exit _______ the endotracheal tube.

A. at the distal end of
B. at the proximal end of
C. at the middle and inside of
D. at the middle and outside of

19. During high frequency jet ventilation (HFJV) or high frequency oscillatory ventilation (HFOV), assessment of a patient's cardiopulmonary status is difficult. Signs of deterioration may include all of the following *except*:

A. respiratory distress.
B. tachycardia.
C. hypotension.
D. pallor.

20. Candidates for HFOV may exhibit all of the following clinical conditions with the *exception* of:

A. difficulty to wean from conventional ventilation.
B. increasing ventilation requirement.
C. rapidly increasing F_IO_2 requirement.
D. chest radiograph consistent with diffuse, homogeneous lung disease.

21. A neonate is being ventilated by a high frequency oscillator. Her physician would like to lower the patient's $PaCO_2$. The therapist should increase the level of ventilation by _______ or _______.

A. increasing the power (amplitude), increasing the frequency (Hz)
B. increasing the power (amplitude), decreasing the frequency (Hz)
C. decreasing the power (amplitude), increasing the frequency (Hz)
D. decreasing the power (amplitude), decreasing the frequency (Hz)

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22. In addition to the F_IO_2 control on a high frequency oscillator, a patient's oxygenation status can be improved by increasing the:

A. power.	C. mean airway pressure.
B. frequency.	D. amplitude.

23. A neonate who is diagnosed with severe RDS has been deteriorating over the past 12 hours. The physician asks a therapist to evaluate this neonate for possible extracorporeal membrane oxygenation (ECMO) therapy. The therapist should *recommend* the neonate for ECMO therapy if she:

A. has a gestational age of more than 34 weeks.
B. weighs less than 2,000 g.
C. has evidence of intracranial hemorrhage (ICH).
D. has been mechanically ventilated for more than 2 weeks.

24. Extracorporeal membrane oxygenation (ECMO) therapy should be considered only when the patient has a(n) _______ percent predicted mortality rate using the conventional ventilator management strategies.

A. 20	C. 60
B. 40	D. 80

25. All of the following may be used to predict an 80% mortality rate under conventional ventilator management strategies *except*:

A. A-a gradient of 605 to 620 mm Hg for 4 to 12 hours.
B. oxygen index of 0.35 to 0.6 for 30 min to 6 hours.
C. PaO_2 of 35 to 50 for 2 to 12 hours.
D. pH of less than 7.25 for 2 hours with hypertension.

26. Which of the following statements is *true* concerning the *venoarterial* route in ECMO therapy?

A. Blood is removed from the right common carotid artery.
B. Blood is removed from the brachial or femoral artery.
C. Blood is returned to the aortic arch via the right common carotid artery.
D. Blood is returned to the aortic arch via the internal jugular vein.

27. Which of the following statements is *true* concerning the *venovenous* route in ECMO therapy?

A. Blood is removed from the right atrium via the right carotid artery.
B. Blood is removed from the right atrium via the femoral vein.
C. Blood is returned to the right atrium via the femoral vein.
D. Blood is returned to the aortic arch via the internal jugular vein.

28. The _______ route in ECMO therapy supports cardiac function because the blood is returned to the _______.

A. venoarterial, aortic arch via the common carotid artery
B. venoarterial, right atrium via the femoral vein
C. venovenous, aortic arch via the common carotid artery
D. venovenous, right atrium via the femoral vein

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Answers to Self-Assessment Questions

1. C.
2. B.
3. A.
4. C.
5. D.
6. A.
7. C.
8. B.
9. C.
10. B
11. A.
12. B.
13. D.
14. A.
15. B.
16. C.
17. A.
18. C.
19. B.
20. A.
21. B.
22. C.
23. A.
24. D.
25. D.
26. C.
27. C.
28. A.

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Additional Resources

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Chapter 18

Mechanical Ventilation in Nontraditional Settings

David W. Chang

Outline

- Introduction
- Mechanical Ventilation at Home
  - *Goals of Home Mechanical Ventilation (HMV)*
  - *Indications and Contraindications*
  - *Patient Selection*
  - *Equipment Selection*
- Mechanical Ventilation in Mass Casualty Incidents
  - *Causes of Mass Casualty*
  - *Mass Casualty and Mechanical Ventilation*
  - *Triage Systems for Mass Casualty Incidents*
  - *Strategic National Stockpile*
  - *Exclusion Criteria for Mechanical Ventilation*
  - *Personnel and Planning*
- Mechanical Ventilation in Hyperbaric Condition
  - *Rationale for Hyperbaric Oxygenation (HBO)*
  - *Indications for HBO*
  - *Endotracheal Tube and Ventilator*
  - *Tidal Volume Fluctuations*
  - *Monitoring and Mechanical Ventilation*
  - *Defibrillation and Cardiac Pacing*
- Mechanical Ventilation in Hypobaric Condition
  - *High-Altitude Cerebral and Pulmonary Edema*
  - *Airplane Cabin Pressure*
  - *Ventilator Parameter Changes under Hypobaric Conditions*
  - *Pressure Compensation*
- Traveling with Portable Ventilators
  - *Characteristics of Portable Ventilators*
  - *Traveling in the United States*
  - *Adjustment of Tidal Volume*
  - *Portable Oxygen Concentrator*
- Summary
- Self-Assessment Questions
- Answers to Self-Assessment Questions
- References
- Additional Resources

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Key Terms

hyperbaric condition
hyperbaric oxygen
hypobaric condition
mass casualty
monoplace hyperbaric chamber
multiplace hyperbaric chamber
pandemic
pressure compensation
SALT
SOFA
START
strategic national stockpile (SNS)
triage

Learning Objectives

After studying this chapter and completing the review questions, the learner should be able to:

- Discuss the indications, contraindications, and patient and equipment selection for mechanical ventilation at home.
- Discuss the use of mechanical ventilation in a mass casualty setting to include the triage systems, strategic national stockpile, exclusion criteria, and personnel and planning.
- List and describe the precautionary measures for mechanical ventilation in a *hyperbaric* condition.
- List and describe the precautionary measures for mechanical ventilation in a *hypobaric* condition.
- List and describe the precautionary measures when using a portable ventilator and oxygen concentrator at high altitudes.

INTRODUCTION

Mechanical ventilation has become more commonplace as technology makes ventilators smaller and more capable for use in nontraditional settings. In addition to home care usage, mechanical ventilators play an important role in mass casualty incidents. Ventilators are also used in hyperbaric medicine for the treatment of ventilator-assisted individuals requiring mechanical ventilation with conditions such as gas gangrene and severe carbon monoxide poisoning. For air transport of critically ill patients, pressure-compensated ventilators for hypobaric conditions are available to reduce fluctuation of delivered volumes. These ventilators are also suitable for ventilator-dependent patients who travel by air.

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MECHANICAL VENTILATION AT HOME

Home mechanical ventilation (HMV) made its first appearance in the United States during the poliomyelitis epidemics of the mid-twentieth century. At that time, negative pressure ventilators (iron lungs) were used to sustain the lives of those who lost the ability to breathe. Today, health care reform and cost containment strategies are limiting the resources available for acute care in the hospitals. Since there are few nursing homes or extended care facilities that will accept the increasing number of ventilator patients, home care becomes an important and viable option for ventilator-dependent patients.

Goals of Home Mechanical Ventilation (HMV)

Mechanical ventilation provided in the home is drastically different from that delivered in an acute care setting. In an acute care setting such as the hospital, the patient is surrounded by an array of medical equipment and supplies. Specialized health care providers are available at all times to provide diagnostic and therapeutic procedures. In addition, the patient in an acute care setting gets little rest because of frequent vital sign assessments and routine laboratory tests. For patients who require long-term mechanical ventilation, it may not be logical or financially feasible to provide mechanical ventilation in an acute care setting.

An alternative to the acute care setting is to provide mechanical ventilation in a nonacute environment such as the patient's home. Three unique and beneficial goals of home ventilator care have been identified as follows (O'Donohue et al., 1986):

1. Extension of the patient's life and enhancement of the quality of life
2. Creation of an environment that will develop and strengthen the patient's physical and physiological functions
3. Reduction of the cost for HMV

Extension of the patient's life is a primary goal of medical and health care procedures. Quality of life is an important issue since a life that has poor quality or little meaning may cause the patient a great deal of anxiety and unnecessary suffering. For this reason, the patient must be involved and be part of the decision-making process before changing the ventilator care plan from an acute care setting to the patient's home.

✔ For patients receiving mechanical ventilation at home, to be able to spend much of the day in a familiar home environment is one benefit that cannot be provided by the hospital.

To be able to spend much of the day in a familiar home environment is one benefit that cannot be provided by the hospital. At home, the patient is likely to become more active in the rehabilitation process. There is an incentive for the patient to try to get well and be weaned off the ventilator. Furthermore, interactions with family members and friends will enhance the patient's psychologic well-being and quality of life.

✔ Reduction of the cost for patient care is another goal of home mechanical ventilation.

Reduction of the cost for patient care is another goal of HMV. The cost savings of providing mechanical ventilation at home can be drastic but should not be the primary consideration. It is vital to ensure that quality care is provided to the patient at or below the cost of patient care in an acute care setting.

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✓ No matter what their background or training, a team approach dedicated and committed to quality care is vital to any successful home ventilator care program.

As we shall discuss later in this chapter, the success of an HMV program requires a team that consists of medical professionals (e.g., physicians, nurses, respiratory therapists, durable medical equipment specialists, dietitians, social workers, and so on) and nonmedical laypersons (e.g., relatives, friends, and support group members). No matter what their background or training, a team approach dedicated and committed to quality care is vital to any successful home ventilator care program (Gower et al., 1985).

Indications and Contraindications

✓ Caretakers involved with HMV must be willing and able to perform the task of taking care of the patient, ventilator, airway, and related medical devices and supplies.

HMV requires a detailed discharge plan because it involves many different agencies, departments, and caretakers. Caretakers involved with HMV must be willing and able to perform the task of taking care of the patient, ventilator, airway, and related medical devices and supplies. For these reasons, the indications for HMV must be clearly defined and they should be based on individual needs.

Indications. Before a decision is made to provide HMV for a patient, four needs-assessment questions should be thoroughly evaluated. The final decision must be based on the answers and solutions to these questions and on the available resources to rectify any remaining patient care issues. The four needs-assessment questions are:

1. Does the patient have a disease state (e.g., high cervical spine injury, severe respiratory muscle paralysis) which may result in persistent ventilatory failure and an inability to be completely weaned from invasive ventilatory support?
2. Does the patient exhibit clinical characteristics (e.g., impending ventilatory failure, cerebral hypoxia) that require mechanical ventilation?
3. Is the patient clinically stable enough to be managed outside an acute care setting?
4. Are there other noninvasive alternatives besides artificial airway and mechanical ventilation (e.g., diaphragm pacing, pneumobelt) suitable for the patient? (AARC CPG, 2007; O'Donohue et al., 1986.)

Diseases That May Benefit from HMV. Lung diseases that may justify HMV outside an acute care setting may be grouped into four categories (Table 18-1). They are:

1. chronic obstructive lung diseases (COPD);
2. restrictive lung diseases;
3. ventilatory muscle dysfunction; and
4. central hypoventilation syndromes (Ferns, 1994; Goldstein et al., 1995; O'Donohue et al., 1986).

Since the severity and coexisting conditions of a disease vary greatly among patients with the same diagnosis, a thorough patient evaluation is a prerequisite for HMV.

✓ HMV should not be initiated if the patient has any unstable medical condition that requires complicated procedures or involves specialized health care personnel.

Contraindications. HMV should not be initiated if the patient has any unstable medical condition that requires complicated procedures or involves specialized health care personnel. Examples may include patients with frequent and progressive arrhythmias,

TABLE 18-1 Diseases That May Benefit from Home Mechanical Ventilation

Pulmonary Problem	Clinical Course
COPD	Airflow obstruction Excessively high compliance Air trapping Acute exacerbation (pneumonia)
Restrictive lung disease	Reduction of lung volumes and capacities Deadspace ventilation Muscle fatigue
Ventilatory muscle dysfunction	Inefficient ventilatory muscle Atelectasis and pneumonia
Central hypoventilation syndrome	Apnea Chronic hypoventilation Atelectasis and pneumonia

and severe oxygen arterial desaturation not responding to low to moderate levels of F_IO_2 or PEEP. Other contraindications may include unwillingness of the patient to receive HMV, and lack of financial and human resources (AARC CPG, 2007).

COPD. COPD is a group of lung impairments that includes chronic asthma, chronic bronchitis, emphysema, and bronchiectasis. Airflow obstruction is the primary clinical feature of these patients. Typically, stable COPD patients require only minimal care such as bronchodilators, flu vaccines, and bronchopulmonary hygiene. Only on rare occasions do they require ventilatory assistance.

✓ Patient with COPD may deteriorate and progress to ventilatory failure as a result of an acute medical condition (e.g., pneumonia) or complications from a major surgical procedure (e.g., abdominal surgery).

COPD patients who require mechanical ventilation are those who develop ventilatory failure, oxygenation failure, or both. On occasion, these patients may deteriorate and progress to ventilatory failure as a result of an acute medical condition (e.g., pneumonia) or complications from a major surgical procedure (e.g., abdominal surgery). When this occurs, blood gases usually reveal acute ventilatory failure (acute respiratory acidosis) superimposed on chronic ventilatory failure (compensated respiratory acidosis). This condition of acute hypercapnia in COPD is also called acute exacerbation of COPD (Malley, 1990). Table 18-2 shows the changes in blood gas results when a stable patient with COPD goes into ventilatory failure and requires mechanical ventilation.

Once placed on a ventilator, COPD patients may be difficult to wean off mechanical ventilation because of inefficient ventilation and sub-optimal gas exchange. This problem is primarily due to airflow obstruction, loss of elastic recoil, and air trapping. In addition, COPD patients usually have coexisting medical problems that are related to the primary lung disease. Some examples of these related medical problems are ventilation/perfusion mismatch, pulmonary hypertension, and cor pulmonale.

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TABLE 18-2 Blood Gas Characteristics of Acute Exacerbation of COPD

Condition	Typical Blood Gases
Chronic ventilatory failure in a stable COPD patient	pH = 7.36, $PaCO_2$ = 55 mm Hg, PaO_2 = 50 mm Hg, HCO_3^- = 30 mEq/L
Acute ventilatory failure in a normal patient due to pneumonia or major surgical procedure	pH = 7.30, $PaCO_2$ = 55 mm Hg, PaO_2 = 75 mm Hg, HCO_3^- = 26 mEq/L
Acute ventilatory failure superimposed on chronic ventilatory failure (e.g., COPD patient with pneumonia)	pH = 7.27, $PaCO_2$ = 74 mm Hg, PaO_2 = 43 mm Hg, HCO_3^- = 33 mEq/L

Patients with COPD also require a high level of care in maintaining the airway. Because of copious amounts of pulmonary secretions and inability to clear secretions effectively, they often require suctioning of the airways and bronchopulmonary drainage. The oxygenation levels also fluctuate widely depending on unanticipated events such as bronchospasm and mucus plugging. Since the weaning process for COPD patients may take days or weeks, home ventilator care becomes a viable option once they are clinically stable and without significant oxygen desaturation. When significant oxygen desaturation (SaO_2 or $SpO_2 < 90\%$) occurs, its cause must be identified and corrected before transferring the patient from hospital to home.

Restrictive Lung Diseases. Restrictive lung diseases such as pulmonary fibrosis and atelectasis limit the patient's ability to expand the lungs. As a result, lung volumes and capacities are reduced. Since minute ventilation requires an adequate tidal volume and respiratory frequency, patients with restrictive lung disease assume a rapid breathing pattern because of the reduction of tidal volume.

The amount of deadspace ventilation is increased in rapid shallow breathing. Furthermore, the work of breathing in restrictive lung diseases is increased because of low lung compliance. High inflation pressure and high respiratory frequency are usually required to maintain adequate ventilation. Over time, these patients develop ventilatory failure secondary to excessive work of breathing and muscle fatigue. Home ventilator care should be considered for patients who have chronic restrictive lung disease and are clinically stable.

Ventilatory Muscle Dysfunction. Patients with ventilatory muscle dysfunction include those afflicted with spinal cord injury or polyneuropathy. Since the primary problem of ventilation is with the ventilatory muscles, they usually have healthy lungs and a good prognosis. Home ventilator care is usually carried out without any complications. Unless there is an infection, these patients generally maintain healthy lungs and normal lung functions. When lung infection occurs, it often leads to pneumonia and atelectasis.

Long-term mechanical ventilation is often necessary for these patients because of the chronic nature of the diseases affecting the respiratory system. These patients

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often recover and do not require prolonged mechanical ventilation except in cases of high spinal injuries (e.g., cervical 1 and 2). Aggressive airway care and bronchopulmonary hygiene should be done to avoid complications resulting from the use of mechanical ventilation and artificial airway.

Central Hypoventilation Syndrome. Patients with central hypoventilation syndrome often exhibit apnea or variable periods of hypoventilation due to failure or dysfunction of the autonomic control of breathing. Because of the range of severity and complications in central hypoventilation syndrome, some patients may require mechanical ventilation only during sleep while others may need it continuously. In patients who have persistent hypoventilation, poor lung expansion may lead to lung infection, atelectasis, and pneumonia. Careful evaluation of these patients in the hospital can help to formulate a care plan for mechanical ventilation in the home.

Patient Selection

Not all patients receiving mechanical ventilation in an acute care setting are suitable candidates for home ventilator care. Typically, a patient who requires a good deal of monitoring and laboratory tests or one who is clinically unstable is ruled out for home ventilator care. Aside from the evaluations based on the medical perspective, four nonmedical factors are crucial in the patient selection process. They are:

1. desires of the patient;
2. desires of the family;
3. cost; and
4. available resources (Eigen et al., 1990; O'Donohue et al., 1986; Smith, 1994).

✓ One disadvantage of leaving the hospital is the feeling of isolation from professional care and the assumption of medical care mainly by family members.

Desires of the Patient. Likely candidates for home ventilator care should be informed about the potential advantages and disadvantages of leaving the hospital. The advantage of being at home is the opportunity for the patient to stay closer to family members and to live in a familiar environment. One disadvantage of leaving the hospital is the feeling of isolation from professional care and the assumption of medical care mainly by family members. The topic of home ventilator care should be discussed when the patient can comprehend the meaning and implications of leaving the acute care setting. It should not take place when the patient is hypoxic, confused, or under emotional distress.

Finally, the decision to implement home ventilator care should not be rushed. Ample time should be provided to the patient so that the decision may reflect the patient's true desire. Hasty discussions and decisions often lead to inaccurate perceptions and poor transition from the hospital to the home care setting.

✓ Successful home ventilator care requires a total commitment from family members and a high level of communication within the family.

Desires of the Family. The desires of family members must be considered because they will be the key persons taking care of the patient and ventilator at home. Depending on the level of ventilator care required by the patient, personal sacrifices must be made. These sacrifices may range from giving up some free time and leisure activities to terminating one's job or career (Smith, 1994). Successful home

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ventilator care requires a total commitment from family members. They must also be able and willing to assume this unfamiliar task of home ventilator care for the duration of time that the patient remains on the ventilator. In one study, the most important factor associated with adaptation of home ventilator care was the level of communication within the family and the degree of commitment between all family members (Glass, 1993).

Cost. The cost of home ventilator care varies greatly and depends primarily on the type of equipment and the extent of professional care required. A ventilator, a backup ventilator, and oxygen supplies are some major equipment expenditures, obtained either by purchase or through a rental agreement. Having medical professionals at home on a regular daily schedule may sometimes be necessary. Depending on the patient's needs, receiving home care provided by nurses and respiratory therapists can be a costly expenditure (Murray, 1989; O'Donohue et al., 1986).

The complexity and available features of a ventilator can affect the equipment cost. As a rule, more complex equipment and supplies cost more. For patients who cannot breathe spontaneously and require continuous ventilatory support, a backup ventilator may also be necessary. Those patients who require around-the-clock care may also need medical professionals to make frequent home visits. Family members may also need to hire a home care aide to provide periodic relief for leisure time and other family or work obligations.

A study on the financial aspects of pediatric home ventilator care shows a significant reduction in the total cost (Hazlett, 1989). Of course, home ventilator care cannot be justified from a financial standpoint if its total cost is higher than the cost of comparable hospital care. Careful cost analysis should be done based on the patient's requirements. The resultant cost for the entire home ventilator care program may be part of the patient selection process.

✔ The primary resources that are vital to the success of home ventilator care include physical environment, technical support, and emotional support.

Available Resources. The primary resources that are vital to the success of home ventilator care include physical resources, technical support, and emotional support. The physical resources for home ventilator care must provide adequate space for the ventilator, a special bed, a wheelchair, oxygen units, and supplies. Suitable and sufficient electrical outlets are also needed for the ventilator, alarms, and other related equipment. Technical support should include one or more home health agencies that have ample equipment and supplies, qualified medical professionals, and around-the-clock coverage. Emotional support may include psychosocial assistance provided to the patient and family members by community agencies, support groups, and friends.

Equipment Selection

Although positive pressure ventilation with an artificial airway is the most common modality in home ventilator care, there are several factors to consider before prescribing the equipment for the patient. The primary factor of equipment selection should result in a ventilator or device that suits the patient's immediate and long-term needs. The secondary factor should deal with the use and maintenance of the equipment.

Types of Ventilatory Support. If the patient does not have adequate spontaneous ventilation for an extended time, positive pressure or negative pressure ventilators are the equipment of choice. Positive pressure ventilation requires an artificial airway, whereas negative pressure ventilation can be provided without an artificial airway. One exception to this practice is the presence of significant airway obstruction.

✔ A backup ventilator may be necessary if the patient is totally dependent on mechanical ventilation.

A backup ventilator may be necessary if the patient is totally dependent on mechanical ventilation. For patients who are using mechanical ventilation on a part-time basis (e.g., during sleep), a backup system may not be needed or may not be financially justifiable.

✔ Other methods of providing ventilatory support include the chest cuirass, raincoat or wrap, pneumobelt, rocking bed, and diaphragmatic pacing.

Other methods of providing ventilatory support include the chest cuirass, raincoat or wrap, pneumobelt, rocking bed, and diaphragmatic pacing. A chest cuirass ventilator is a shell that fits over the patient's chest wall (Figure 18-1). The raincoat or wrap is an airtight jacket that seals at the arms, hips, and neck. It covers a larger area than the chest cuirass and does not impinge on the chest and abdomen. For this reason, it offers a larger inspiratory volume to the user. The raincoat or wrap is more difficult to get into and usually requires help from another person in the home. The pneumobelt is a corsetlike belt attached to a positive pressure generator. The positive pressure inflates the belt, squeezes the abdomen, and pushes the diaphragm upward. An alternating sequence of positive pressure and ambient pressure provided to the pneumobelt produces ventilation. The rocking bed relies on motion to displace the abdominal contents to facilitate diaphragmatic motion and ventilation (Hill, 1994; Votroubek, 1995).

Diaphragmatic Pacing. Diaphragmatic pacing or bilateral pacing of the phrenic nerves does not actually provide mechanical ventilation. Rather, it is used to augment spontaneous ventilation. Diaphragmatic pacing has been used in infants and children for more than a decade (Ilbawi et al., 1985). The pacing system includes an external transmitter and antenna placed on the skin over a receiver implanted subcutaneously. At a predetermined interval, the receiver sends electrical energy to an electrode placed near the thoracic phrenic nerves. When the phrenic nerves are stimulated by electrical energy, the diaphragm contracts (Votroubek, 1995).

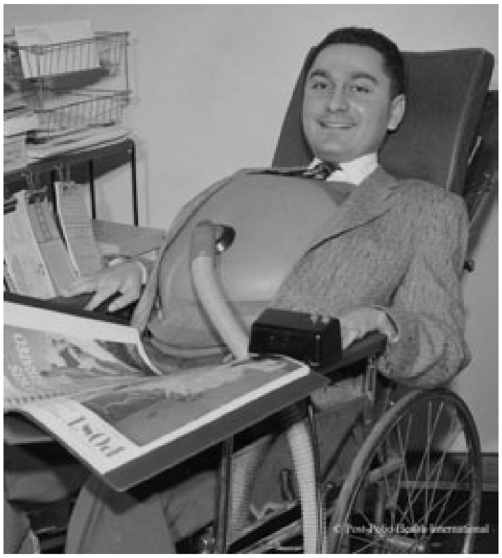
Courtesy of Post-Polio Health International

FIGURE 18-1 Chest cuirass in use by a man following the polio epidemic in the 1950's.

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✓ The ventilator used at home should be highly dependable and should require infrequent or no maintenance by nonmedical personnel such as the family members and home care assistants.

Reliability and Safety. The ventilator used at home should be highly dependable and should require infrequent or no maintenance by nonmedical personnel such as the family members and home care assistants. Each ventilator should have safety features such as high pressure, low pressure, and ventilator failure alarms. These features must not be too complicated for those working with the patient and ventilator.

Simplicity and Portability. The operation and maintenance of home care ventilators should be direct and simple. Dials and alarms on the ventilator that are illogical and hard to understand are likely to confuse the users. The ventilator circuits and supplies should be disposable or simple to clean and disinfect if they are reusable.

Ventilators that are small, compact, and portable will provide the most flexibility when the patient wants to move around the home. Ventilators with built-in rechargeable battery packs are also very versatile in the event of brief power failure. The patient may also take advantage of this portability feature to make physician office visits or brief shopping trips.

In summary, a successful HMV program demands proper patient selection, careful planning, detailed home instruction, and a programmatic follow-up by the health care team. The team approach is probably the most critical element of successful ventilator care in the home. The health care professionals, and especially the family members, must be able to make a long-term commitment in caring for the mechanically ventilated patient at home.

MECHANICAL VENTILATION IN MASS CASUALTY INCIDENTS

mass casualty: A large number of severely injured or deaths that exceeds a timely response from regional support centers.

Mass casualty refers to a large number of severely injured or deaths that exceeds a timely response from regional support centers. Mechanical ventilation is a necessity in the management of victims of mass casualty, as they often suffer from head and chest trauma, ARDS, and ventilatory failure.

Causes of Mass Casualty

✓ Mass casualty incidents may be due to natural (e.g., earthquakes, pandemics) or man-made events (e.g., terrorism, wars).

Mass casualty incidents may be due to natural or man-made events. Natural events such as Tsunamis, earthquakes, and pandemics have caused mass casualty incidents with high mortalities.

Man-made events that can cause mass casualty include war, terrorism, acts of civil disobedience, bomb explosions, and exposure to radiologic and chemical agents (e.g., Bhopal, India, 1984).

These events have caused millions of casualties; many of the victims died from conditions leading to ventilatory failure.

Natural and Man-Made Causes. Throughout history, natural disasters have caused many casualties. The Indian Ocean tsunami on December 26, 2004, caused up to 225,000 deaths (MMWR, 2005), and 127,000 people were listed as missing (CNN, 2005). In the Haiti earthquake on January 12, 2010, the Haiti government estimated more than 215,000 were killed and 300,000 injured as a result of the earthquake (Ryan, 2010). Casualties caused by man are common in prolonged and large-scale wars throughout history. In World War II, estimates of total deaths range from 50 million to 70 million. Estimated civilian casualties range from 40 to 52 million, including 13 to 20 million from war-related disease and famine. Total military casualties ranged from 22 to 25 million, including deaths in captivity of about 5 million prisoners of war (Roberts, 2011; Wikipedia, 2011). On a smaller scale but with equal devastation, the simultaneous bombing of four commuter trains in Madrid is an example of a man-made mass casualty incident. The bombings on March 11, 2004, killed 177 people and injured more than 2,000. Many of these victims suffered primary (and compressive) blast injury to the lungs and required mechanical ventilation, fluid management, and supportive care (Hunt, 2010).

✓ Victims of primary blast injury to the lungs often require mechanical ventilation, fluid management, and supportive care.

National and international events increase the utilization of health care for those who have been exposed to chemical agents such as nerve agents, blood agents, choking agents, riot-control, and paralyzing agents (e.g., Moscow hostage crisis, October 28, 2002). Among these agents, nerve agents often require mechanical ventilation. Nerve agents are acetylcholinesterase inhibitors that lead to an accumulation of acetylcholine at the muscarinic and nicotinic receptors throughout the body. Exposure to large amounts of nerve agents (a sudden surge of acetylcholine) may quickly induce loss of consciousness, seizures, flaccid paralysis, and apnea (McCafferty, 2002).

✓ Nerve agents used in the war or terrorism acts are acetylcholinesterase inhibitors that lead to accumulation of acetylcholine at the muscarinic and nicotinic receptors throughout the body. A sudden surge of acetylcholine may quickly induce loss of consciousness, seizures, flaccid paralysis, and apnea.

Pandemics. A **pandemic** is the occurrence of infectious disease that is spreading through human populations across a large region, continent, or the world. Infectious diseases have been the primary cause of mass casualty incidents throughout history. Some notable ones are the Black Death and Spanish Flu. The Black Death pandemic (bacterium *Yersinia pestis*) killed between 75 and 100 million people around the world in 1400 (Wikipedia, 2010). From March 1918 to June 1920, the Spanish Flu pandemic (influenza A/H1N1) caused 50 to 100 million deaths worldwide (Johnson, 2002). Recent H1N1 outbreaks in 2009 showed a much lower mortality rate due to a better knowledge base of the disease and quick implementation of isolation procedures. Among those patients with confirmed cases, many developed acute lung injury or acute respiratory distress syndrome and required mechanical ventilation (Rello et al., 2009; Venkata et al., 2010). Management modalities for some of these patients included inhaled nitric oxide, high frequency oscillatory ventilation, extracorporeal membrane oxygenation, and prone positioning (Kumar et al., 2009; MMWR, 2009).

pandemic: An occurrence of infectious disease that is spreading through human populations across a large region, continent, or world.

✓ Among those patients with confirmed cases of H1N1, many developed acute lung injury or acute respiratory distress syndrome and required mechanical ventilation.

Mass Casualty and Mechanical Ventilation

In 2005, the U.S. Department of Health and Human Services estimated the number of health care utilization and deaths associated with moderate and severe pandemic influenza scenarios (Table 18-3). The number of mechanical ventilators that would be required for a 1958/68-like outbreak is estimated to be 64,875. For an influenza outbreak on the scale of 1918, an estimated 742,500 ventilators would be needed (HHS, 2005). Based on a study in 2006, there were 105,000 ICU ventilators available in the U.S. Among these ventilators, about 100,000 would be in use

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TABLE 18-3 Projection of Health Care and Mechanical Ventilation Utilization in Moderate and Severe Pandemic Influenza Scenarios*

Characteristic	Moderate (1958/68-like)	Severe (1918-like)
Illness	90 million (30% of population)	90 million (30% of population)
Outpatient medical care	45 million (50% of illness)	45 million (50% of illness)
Hospitalization	865,000	9,900,000
ICU care	128,750	1,485,000
Mechanical ventilation	64,875	742,500
Deaths	209,000	1,903,000

**Projection based on extrapolation from past pandemics in the United States. These estimates do not include the potential positive impact of medical and nonmedical interventions that were not available in the past pandemics (HHS, 2005).*

at any given time (McNeil, 2006). The availability of the remaining 5,000 ventilators for the entire U.S. represents a critical shortage in ventilators and qualified personnel to manage the surge in demand.

Triage Systems for Mass Casualty Incidents

Triage can be defined as a process using predetermined criteria to assign individuals from a large pool of people for grouping and making decisions. The principles of triage were used as early as during Napoleon's wars (1799–1815). Earlier military triage placed a high priority on *less wounded* soldiers who could be returned to the battlefield (Mitchell, 2008).

triage: A process that uses predetermined criteria to assign individuals from a large pool of people for grouping and making decisions.

Today, triage puts a high priority of care on those *severely ill* individuals who are most likely to survive. There are many triage systems and algorithms (Lerner, 2008), and three representative systems are discussed in the following sections.

Prehospitalization triage systems. The most widely used prehospitalization triage algorithm in the U.S. is the **START** (***S**imple **T**riage **a**nd **R**apid **T**reatment*) system. START is a simple triage and rapid treatment algorithm suitable for use by first responders. It is based on three parameters: respiration, perfusion, and mental status (RPM). The pediatric version is known as JumpSTART.

START: the Simple Triage and Rapid Treatment algorithm that is based on three parameters: respirations, perfusion, and mental status (RPM). The pediatric version is known as JumpSTART.

✓ The pediatric version of START is known as JumpSTART.

START was developed by the Newport Beach Fire and Marine Department and Hoag Hospital in Newport Beach, California in 1983. Modifications to START were made in which radial pulse substituted the capillary refill time. Figure 18-2 shows the algorithm of the START triage system (Benson et al., 2006; cert-la.com, 2011).

While START is one of the most common triage systems, there is limited scientific *evidence* to validate the effectiveness or accuracy of START or other triage systems. In 2008, a multidisciplinary committee reviewed 9 existing mass casualty triage systems (7 adult- and 2 pediatric-specific systems), and it used the best available

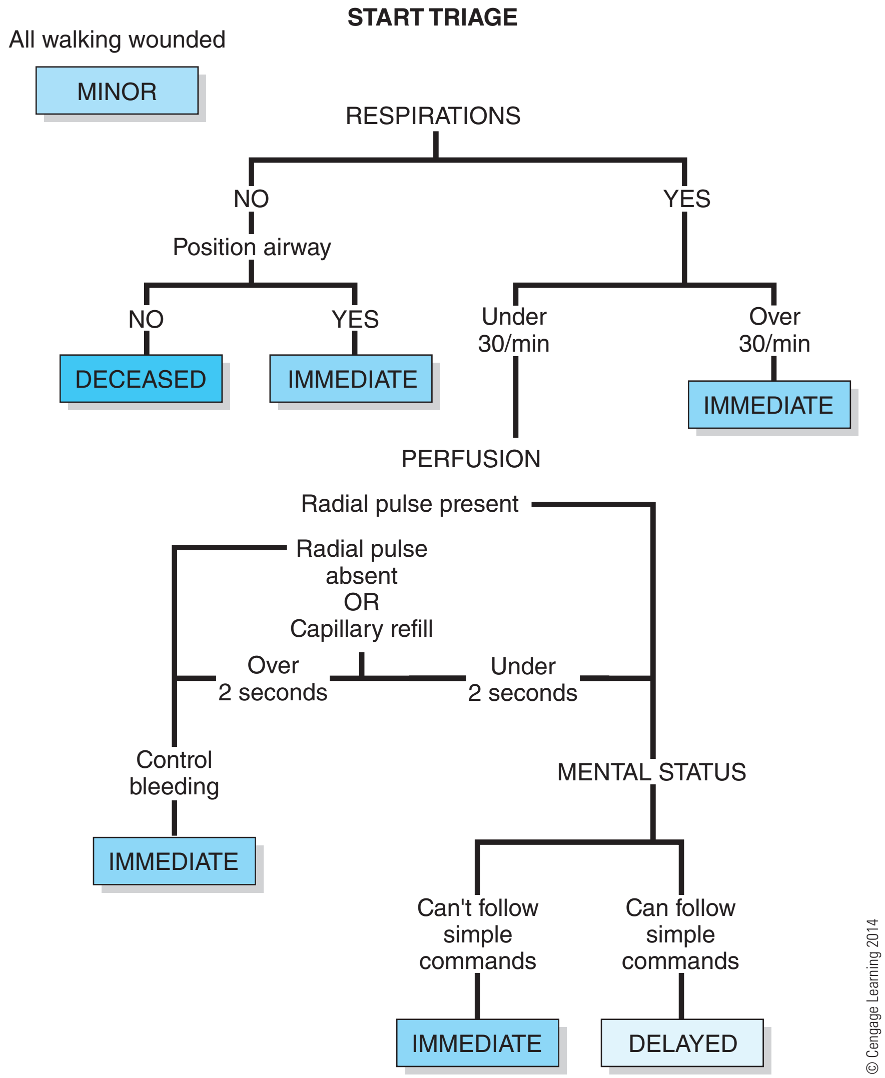
START TRIAGE
All walking wounded
MINOR
RESPIRATIONS
NO
Position airway
NO
DECEASED
YES
IMMEDIATE
YES
Under 30/min
Over 30/min
IMMEDIATE
PERFUSION
Radial pulse present
Radial pulse absent OR Capillary refill
Over 2 seconds
Under 2 seconds
Control bleeding
IMMEDIATE
MENTAL STATUS
Can't follow simple commands
IMMEDIATE
Can follow simple commands
DELAYED
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FIGURE 18-2 START stands for Simple Triage and Rapid Treatment. Immediate has a highest priority of care than Delayed and Minor.

✔ The **SALT** triage algorithm (Figure 18-3) incorporates triage with life-saving interventions (LSI): control of major hemorrhage, opening of airway, chest decompression, and auto injector antidotes.

SALT: a mass casualty triage algorithm consists of Sort, Assess, Life-saving interventions, and Treatment/Transport.

SOFA: the Sequential Organ Failure Assessment (SOFA) score is a triage system that uses six criteria to predict the outcomes of critically ill patients in the hospital.

✔ The SOFA scoring system is developed to assess hospitalized patients. For patients presenting with sepsis or evidence of hypoperfusion, SOFA may be an appropriate assessment tool for initial and ongoing ICU assessments. It may serve as a predictive and prognostic model for critical care clinical outcomes.

scientific information and developed SALT (Lerner, 2008). **SALT** (Sort, Assess, Life-saving interventions, Treatment/Transport) is a mass casualty triage algorithm (Figure 18-3) that incorporates triage (sort) with immediate life-saving interventions (LSI). LSI includes control of major hemorrhage, opening of airway, chest decompression, and auto injector antidotes. Like START and JumpSTART, SALT is used as an in-the-field (prehospitalization) triage system.

Triage System for Hospitalized Patients. The Sequential Organ Failure Assessment (**SOFA**) score (mdcalc, 2011) in Table 18-4 is a triage system for hospitalized patients. It was created in a consensus meeting of the European Society of Intensive Care Medicine in 1994, and further refinements were made to it in 1996. SOFA uses six criteria to predict the outcomes of critically ill patients in the hospital (Ferreira et al., 2001; Jones et al., 2009). The SOFA scores are used to prioritize patient care based on available resources. For patients presenting with sepsis or evidence of

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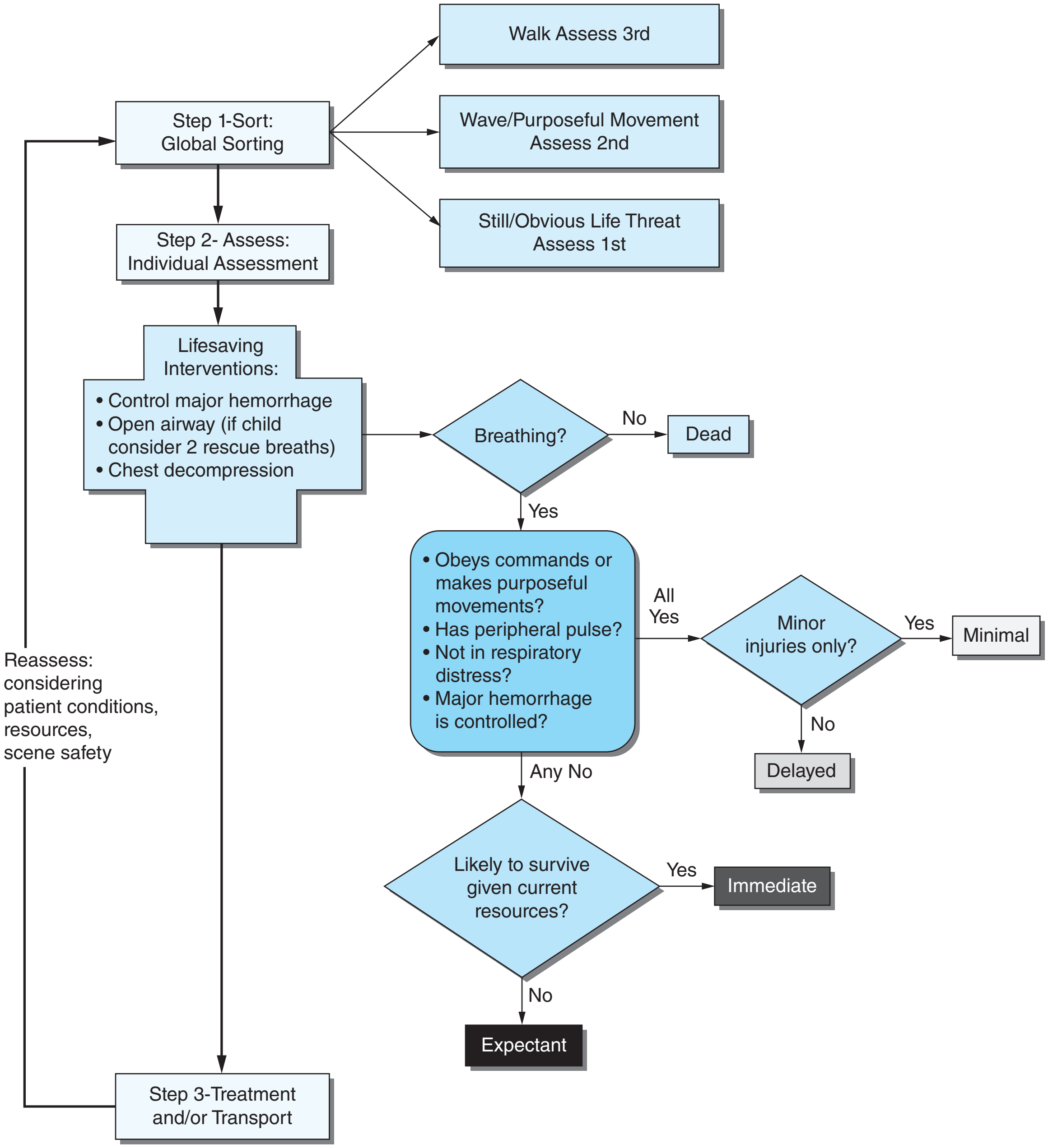
Walk Assess 3rd
Step 1-Sort: Global Sorting
Wave/Purposeful Movement Assess 2nd
Still/Obvious Life Threat Assess 1st
Step 2- Assess: Individual Assessment
Lifesaving Interventions:
• Control major hemorrhage
• Open airway (if child consider 2 rescue breaths)
• Chest decompression
Breathing?
No
Dead
Yes
• Obeys commands or makes purposeful movements?
• Has peripheral pulse?
• Not in respiratory distress?
• Major hemorrhage is controlled?
All Yes
Minor injuries only?
Yes
Minimal
No
Delayed
Reassess: considering patient conditions, resources, scene safety
Any No
Likely to survive given current resources?
Yes
Immediate
No
Expectant
Step 3-Treatment and/or Transport

FIGURE 18-3 SALT (Sort, Assess, Life-savings interventions, Treatment/Transport) algorithm for mass casualty triage. Expectant has the highest priority of care than Immediate, Delayed, and Minimal.

Source: Radiation Emergency Medical Management, U.S. Department of Health and Human Services. Accessed June 29, 2012. http://www.remm.nlm.gov/salttriage.htm

hypoperfusion, SOFA may be an appropriate assessment tool for initial and ongoing critical care assessments. It may also serve as a predictive and prognostic model for critical care clinical outcomes (Jones et al., 2009).

The parameters used in the SOFA scoring system are related to oxygenation, blood clotting, liver function, blood pressure, neurologic function, and kidney function. A high score (e.g., ≥11) calls for palliation care only as it correlates with a mortality rate of 95%. A SOFA score from 10 to 8 calls for the highest priority of critical care. Intermediate critical care is provided to those with scores of ≤7 (Ferreira et al., 2001).

✓ A high SOFA score (e.g., ≥11) calls for palliation care only as it correlates with a mortality rate of 95%. A SOFA score from 10 to 8 calls for the highest priority of critical care.

TABLE 18-4 Sequential Organ Failure Assessment (SOFA) Score

Variable/Score	0	1	2	3	4
Criteria/Score	0	1	2	3	4
Respiratory PaO_2/F_IO_2 (mm Hg)	>400	≤400	≤300	≤200	≤100
Coagulation Platelets, × 103/μL (× 106/L)	>150 (>150)	≤150 (≤150)	≤100 (≤100)	≤50 (≤50)	≤20 (≤20)
Liver Bilirubin, mg/dL (μmol/L)	<1.2 (<20)	1.2–1.9 (20–32)	2.0–5.9 (33–100)	6.0–11.9 (101–203)	>12 (>203)
Cardiovascular Hypotension (infusion dosages in μg/kg/min)	None	MAP <70 mmHg	Dop ≤ 5 or dobutamine any dose	Dop >5, Epi ≤ 0.1, or Norepi ≤ 0.1	Dop > 15, Epi > 0.1, or Norepi >0.1
Central Nervous System Glasgow Coma Score	15	13–14	10–12	6–9	<6
Renal Creatinine, mg/dL (μmol/L) or urine output (use worst value)	<1.2 (<106)	1.2–1.9 (106–168)	2.0–3.4 (169–300)	3.5–4.9 (301–433) or urine output <500 mL/day	>5 (> 434) or urine output <200 mL/day

Dopamine [Dop], epinephrine [Epi], norepinephrine [Norepi]. SI units in brackets.
Reference http://www.mdcalc.com/sequential-organ-failure-assessment-sofa-score.

Strategic National Stockpile

✓ The SNS program owns and maintains approximately 10,000 mechanical ventilators for distribution to states affected by mass casualty.

strategic national stockpile (SNS): a federal program that stores large quantities of medicine and medical supplies in a centralized location for the public in case of a health emergency.

The Centers for Disease Control and Prevention (CDC) uses a **strategic national stockpile (SNS)** program to store large quantities of medicine and medical supplies in a centralized location for the public in case of a health emergency. The SNS program owns and maintains approximately 10,000 mechanical ventilators for distribution to states affected by mass casualty (AARC, 2006; news medical, 2009). There are advantages and disadvantages of a stockpile program where all of the medicines and medical supplies are stored at a centralized location managed by the federal government. Pros include the immediate delivery of a large number of ventilators to hospitals experiencing an isolated disaster. Cons include the potential lack of experience and troubleshooting skills by clinicians with the brand-specific

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✓ The LP-10 (discontinued in November of 2006) and Uni-Vent Eagle 754 are being replaced with the LTV 1200 (CareFusion) and Newport HT50 portable ventilators.

ventilators in the stockpile (Wilgis, 2008). At the state level, each state has plans to distribute the SNS medicines and medical supplies to local communities as quickly as possible (CDC, 2010).

Since the beginning of the stockpile program for mechanical ventilators, the LP-10 and Uni-Vent Eagle 754 had been the two ventilators designated by the federal government for mass casualty incidents (Malatino, 2008). The LP-10 (discontinued in November of 2006) and Uni-Vent Eagle 754 are being replaced with the LTV 1200 (CareFusion) and Newport HT50 portable ventilators. Readers may refer to the manufacturer websites to obtain the respective technical specifications and operating manuals. Other than the ventilators in the federal SNS program, there are other portable ventilators that may be suitable for use in mass casualty incidents (e.g., EPV 100, Allied Healthcare Products, Inc.).

Exclusion Criteria for Mechanical Ventilation

✓ The NYS DOH exclusion criteria are based on objective clinical conditions and do not rely on ethical or quality-of-life issues. Individuals meeting these exclusion criteria would not be placed on a ventilator should the number of ventilators fail to meet the surge in demand.

The number of ventilators in the Strategic National Stockpile is clearly insufficient for the projected number of people needing mechanical ventilation in a major mass casualty incidence (AARC, 2006; HHS, 2005). Exclusion criteria for ventilator access have been developed to prioritize the allocation of a limited number of ventilators in the event of a mass casualty incident.

For example, the New York State Department of Health (NYS DOH) outlined the exclusion criteria for ventilator access (NYS DOH, 2007). The exclusion criteria are based on objective clinical conditions and do not rely on ethical or quality-of-life issues. They focus on a person's history of cardiac arrest, presence of metastatic malignancy, severe burn, and organ failure (Table 18-5). Individuals meeting these exclusion criteria would not be placed on a ventilator should the number of ventilators fail to meet the surge in demand.

TABLE 18-5 Exclusion Criteria for Ventilator Access (NYS DOH)

1. Cardiac arrest: unwitnessed arrest, recurrent arrest, arrest unresponsive to standard measures, trauma-related arrest
2. Metastatic malignancy with poor prognosis
3. Severe burn: body surface area >40%, severe inhalation injury
4. End-stage organ failure:
   Cardiac: NY Heart Association class III or IV
   Pulmonary: severe chronic lung disease with $FEV_1 < 25\%$
   Hepatic: MELD (model of end-stage liver disease) score >20
   Renal: dialysis-dependent
   Neurologic: severe, irreversible neurologic event/condition with high expected mortality

Reference NYS DOH (2007).

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Allocation of Ventilators. The Centers for Disease Control and Prevention (CDC, 2011) published an article that describes the biomedical and ethical principles for the allocation of mechanical ventilators. Topics in the article include: respect for persons and their autonomy, beneficence, justice, maximizing net benefits, social worth, life cycle principle, fair chances versus maximization of best outcomes, and who should make ventilator allocation decisions. Readers should refer to this article for a detailed discussion and additional resources.

Personnel and Planning

✓ The Department of Homeland Security developed the Homeland Security Exercise and Evaluation Program (HSEEP) to assist state and local governments to develop, implement, and evaluate training exercise programs to enhance preparedness.

In addition to personnel providing direct medical care, other personnel and services are required during mass casualty incidents. They include transportation and evacuation, infection and vector control, security, volunteer management, worker health and safety, pharmacy, social service, utility outage, wastewater and solid waste disposal, nutrition, blood products and services, food safety and security, public health and medical information, veterinary services, victim identification, and mortuary services. The Department of Homeland Security developed the Homeland Security Exercise and Evaluation Program (HSEEP) to assist state and local governments to develop, implement, and evaluate training exercise programs to enhance emergency preparedness (Macintyre, 2009; CNA Corporation, 2004).

MECHANICAL VENTILATION IN HYPERBARIC CONDITION

hyperbaric oxygen: supplemental oxygen under hyperbaric conditions; used in conditions such as severe carbon monoxide poisoning, decompression sickness, gas gangrene, and anaerobic infections.

hyperbaric condition: an environment in which the atmospheric pressure is greater than one barometric pressure (e.g., diving under water, hyperbaric chamber).

multiplace hyperbaric chamber: a large hyperbaric chamber designed to treat more than one patient at a time.

Hyperbaric oxygen (HBO) refers to oxygen therapy administered under a **hyperbaric condition**. It has been in use extensively for conditions such as severe carbon monoxide poisoning, decompression sickness, gas gangrene, and anaerobic infections (Vazquez et al., 2003). In some patients, intensive care support and mechanical ventilation are necessary during HBO therapy. This section reviews the common issues when mechanical ventilation is used in a **multiplace hyperbaric chamber**.

Rationale for Hyperbaric Oxygenation (HBO)

Hyperbaric oxygenation increases the amount of dissolved oxygen carried by the plasma. At an F_IO_2 of 21%, the normal oxygen content is 20 vol% and the plasma carries about 0.3 vol% of the total oxygen content.

✓ Tissues require a minimum of 60 mL of oxygen per liter (or 6 vol%) of blood flow to maintain normal metabolism in cells and many tissues.

Tissues require a minimum of 60 mL of oxygen per liter (60/1,000 = 6/100 or 6 vol%) of blood flow to maintain normal metabolism (Leach et al., 1998). At 1 atmospheric pressure (760 mm Hg at sea level) and at an F_IO_2 of 100%, the PaO_2 is about 673 mm Hg and the calculated dissolved oxygen is about 2 vol%. At 3 atmospheres, the dissolved oxygen is 6 vol% or 6 mL per 100 mL of blood. This amount of oxygen meets the minimal requirement for normal cellular metabolism.

At an F_IO_2 of 100%,

$$
\begin{aligned}
P_AO_2 &= (P_B - P_{H_2O}) \times F_IO_2 - PaCO_2 \\
&= (760 - 47)\text{ mm Hg} \times 100\% - 40\text{ mm Hg} \\
&= 673\text{ mm Hg}
\end{aligned}
$$

Since $PaO_2 \approx P_AO_2$

$$
\begin{aligned}
\text{Dissolved oxygen at 1 ATA} &= P_AO_2 \times 0.003 \\
&= (673 \times 0.003)\text{ vol\%} \\
&\approx 2\text{ vol\%} \\
\text{Dissolved oxygen at 3 ATA} &= 3 \times 2\text{ vol\%} \\
&\approx 6\text{ vol\%}
\end{aligned}
$$

Indications for HBO

One barometric pressure equals 760 mm of mercury or 33 ft of water. At 33 ft below the sea level, the total barometric pressure is 1,520 mm Hg (760 mm Hg × 2). Decompression sickness occurs when a diver surfaces too rapidly. Rapid decompression of pressure causes the dissolved gases in blood to form gas bubbles. These bubbles can migrate to different parts of the body, causing moderate to severe adverse outcomes, ranging from joint pain to paralysis and death. High-altitude decompression sickness can also occur upon rapid ascent from low altitude to high altitude over 18,000 ft (Leach et al., 1998). Treatment for both conditions includes recompression quickly in a hyperbaric chamber followed by *gradual* decompression.

Treatment for diver decompression and high-altitude decompression includes recompression quickly in a hyperbaric chamber followed by *gradual* decompression.

Patients with severe carbon monoxide (CO) poisoning often benefit from HBO. CO poisoning reduces the oxygen-carrying capacity of hemoglobin, and HBO compensates for this deficiency by increasing the amount of dissolved oxygen. The symptoms of severe CO poisoning include unconsciousness, convulsions, and neurological impairment. Mild to moderate CO poisoning may be treated with 100%-inspired oxygen (via a non-rebreathing mask) until the carboxyhemoglobin (COHb) level is less than 5% (Boutros, 1976). In severe CO poisoning, HBO is needed to treat acute tissue hypoxia (Leach et al., 1998).

Mild to moderate CO poisoning may be treated with 100%-inspired oxygen until the COHb is less than 5 vol%.

Another indication for HBO is myonecrosis and gas gangrene of soft tissues due to clostridial infection. These anaerobic infections (e.g., *Clostridium perfringens*) respond to HBO in some cases because an oxygen-enriched environment hinders the growth of anaerobic pathogens. HBO is usually used as an adjunctive therapy to antibiotics, along with abridgement of necrotic soft tissues (Leach et al., 1998).

Another indication for HBO is myonecrosis and gas gangrene of soft tissues due to anaerobic clostridial infection.

Endotracheal Tube and Ventilator

For patients requiring an endotracheal tube in the hyperbaric chamber, water (instead of air) is used to filled the cuff of the endotracheal tube. A fluid-filled cuff prevents overdistension and rupture of the cuff during decompression. Neuromuscular blocking agents have a shorter half-life under hyperbaric conditions. The degree of block must be monitored closely. If necessary, the blocking agents need to be administered at lower dosages but more frequently (Desola, 1988).

A fluid-filled ET tube cuff prevents overdistension and rupture of the cuff during decompression in a hyperbaric chamber.

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TABLE 18-6 Selected Ventilators Suitable for Use in Multiplace Hyperbaric Chambers

Ventilator	Testing Conditions and Notes
Bird Avian (Bird Technologies, Palm Springs, USA)	VCV only. Tested to 2.5 ATA.
EVITA 4 (Dragerwerk, Germany)	VCV and PCV. Tested to 2.8 ATA also in CPAP and PSV modes.
Impact Uni-Vent Eagle Model 754 (Impact Instrumentation, Inc., New Jersey, USA)	VCV only. Tested to 6 ATA.
Lifecare PLV-100	VCV and PCV. Tested to 6 ATA also in SIMV and assist modes.
Omni-Vent Series D (Allied Healthcare Products, Inc., California, USA)	VCV only. Tested to 6 ATA.
Servo 900 C (Siemens-Elema, Sweden)	VCV and PCV. Tested to 6 ATA.

Reference: Kot, 2006.

✓ Pressure-controlled ventilation provides a constant pressure and delivers more stable tidal volumes during compression and decompression.

Pressure-controlled ventilation (PCV) is preferred when mechanical ventilation is required in a multiplace hyperbaric chamber. Since PCV provides a constant pressure, it delivers more stable tidal volumes. Volume-controlled ventilation (VCV) requires frequent adjustments of the tidal volume during compression and decompression (Kot, 2006).

There are many ventilators that can be used safely under hyperbaric conditions. The ventilators in Table 18-6 offer different modes of ventilation and are capable of operating under various barometric pressures (Kot, 2006).

Tidal Volume Fluctuations

Boyle's Law describes the inverse relationship between pressure and volume. In hyperbaric conditions, pressure causes compression of gases and reduction of gas volume. In volume-controlled ventilation under hyperbaric conditions, the delivered tidal volume is therefore less than the set tidal volume. As the pressure increases in the multiplace hyperbaric chamber, the set tidal volume should be increased to compensate for the effects of gas compression. In mechanical ventilation, the expired volume approximates the delivered volume. A mechanical respirometer (e.g., Wright respirometer) may be used to monitor the expired tidal volume during gas compression and decompression. The tidal volume setting can be adjusted using the measured tidal volume as a guide.

✓ In mechanical ventilation, the expired volume approximates the delivered volume. A mechanical respirometer (e.g., Wright respirometer) may be used to monitor the changing tidal volume during gas compression and decompression.

In one study, a mechanical respirometer is used to monitor the volume changes of a ventilator during compression. To compensate for the gas compression, a progressive increase of set volumes is needed to maintain stable minute and tidal volumes. At 2.8 ATA pressure, a set tidal volume of 710 mL is needed to deliver a tidal volume of 500 mL (Table 18-7) (Vazquez et al., 2003).

Ventilators operating under hyperbaric conditions should be evaluated for changes in pressure, frequency, and other electronic controls. Adjustments of these controls

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TABLE 18-7 Volume Changes to Maintain Stable Delivered Volumes at Frequency of 12/min

ATA Pressure	Set V_T (mL)	Delivered V_T (mL)	Delivered V_E (L/min)
1	500	500	6000
1.3	550	500	6000
1.6	600	500	6000
1.9	650	500	6000
2.2	675	500	6000
2.5	695	500	6000
2.8	710	500	6000

Reference: Vazquez et al., 2003.

and settings are necessary during compression and decompression in order to maintain the desired tidal volume and minute ventilation.

Monitoring and Mechanical Ventilation

Special precautions and safeguard procedures are necessary for mechanically ventilated patients undergoing treatment in the hyperbaric chamber. Since some medical equipment or instruments use electricity or create voltaic arc, their structure and functions must be checked for safety due to the presence of high pressure and electricity in an oxygen-enriched environment. Medical equipment and devices used in a hyperbaric environment must be tested prior to patient application. Table 18-8 lists some common noninvasive cardiopulmonary monitoring devices that have been used successfully in a hyperbaric environment (Kot, 2005).

Since some medical equipment or instruments use electricity or create voltaic arc, their structure and functions must be checked for safety due to the presence of high pressure and electricity in an oxygen-enriched environment.

For invasive intravascular monitoring, systemic and pulmonary pressures (e.g., BP, CVP, PAP, PCWP) can be measured safely, provided that the transducer is also placed under hyperbaric condition, is properly filled with fluid, and is correctly calibrated. With proper procedure and equipment, cardiac output, intracranial pressure, and blood gas analysis are some other invasive procedures that can be performed safely under hyperbaric condition (Kot, 2005).

systemic and pulmonary pressures (e.g., BP, CVP, PAP, PCWP) can be measured safely, provided that the transducer is also placed under hyperbaric condition, is properly filled with fluid, and is correctly calibrated.

Defibrillation and Cardiac Pacing

Defibrillation can be done in a multiplace chamber provided that several conditions are met (see Table 18-9).

Defibrillation is a dangerous procedure in the hyperbaric chamber because of the likelihood of fire caused by electrical discharges and voltaic arc between the paddles. It should not be done in a **monoplace hyperbaric chamber** (Figure 18-4) due to its limited space to carry out safety precautions. However, defibrillation

monoplace hyperbaric chamber: a hyperbaric enclosure (e.g., cylindrical metal or acrylic crystal clear tube) designed to treat one patient at a time.

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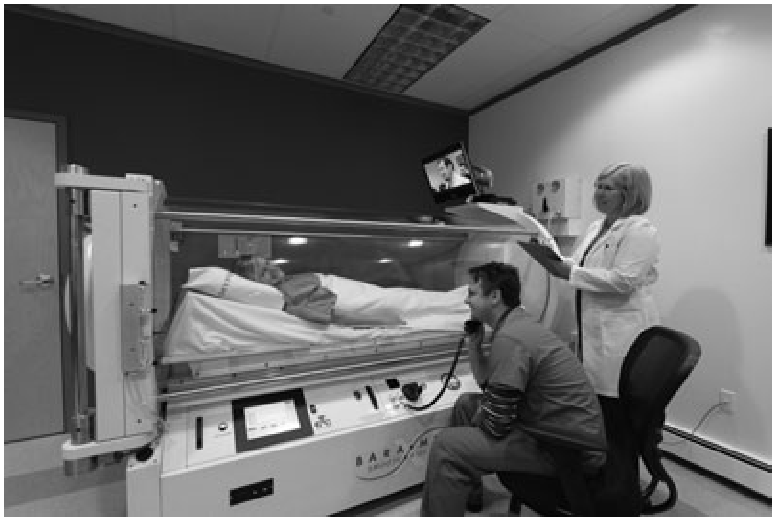
TABLE 18-8 Use of Noninvasive Cardiopulmonary Monitoring Devices in a Hyperbaric Chamber

Noninvasive Cardiopulmonary Monitoring Device	Special Consideration
ECG	ECG signals are transferred outside the hyperbaric chamber for monitoring
Pulse oximetry	Use is limited to pulse rate only because HBO usually results in full oxygen saturation for patients without cardiovascular deficits
Blood pressure	Manual method is preferred as automated (electrical) method may pose fire hazards
Temperature	Electrical thermistors should be safe Mercury-based device should not be used due to toxicity of mercury under pressure
$TcPO_2$ and $TcPCO_2$	Suitable for use in hyperbaric chamber to monitor oxygenation and ventilation
$P_{ET}CO_2$	Mainstream device may provide false high results due to increase in gas density Sidestream device is preferred to monitor end-tidal PCO_2 outside the hyperbaric chamber using decompressed expiratory tubing
Spirometry and airway pressure	Electric spirometer (e.g., Ohmeda 5420) may be used to monitor airway pressure and exhaled tidal volume Mechanical respirometer (e.g., Wright) can be used to monitor exhaled tidal volume

Reference: Kot, 2005.



Courtesy of Environmental Teclonics Corporation

FIGURE 18-4 Monoplace hyperbaric chamber.

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TABLE 18-9 Safety Requirements for Defibrillation in a Multiplace Hyperbaric Chamber

Safety Requirement	Notes
Multiplace chamber is compressed with air and F_IO_2 is kept below 21.5%	Room air reduces risk of fire
Large adhesive plates are attached to the patient's chest and gel is applied to provide conduction	Flammable materials must not be around the large adhesive plate
Transmission cable of large diameter and low resistance goes through the chamber wall and resides outside the chamber	Special cable reduces electrical load during defibrillation
Defibrillator and switches are located outside the chamber	Discharge unit outside the chamber eliminates electrical discharges inside the chamber
A three-person team should be available as follows: (1) one person attaching paddle inside the chamber, (2) one person operating the discharge unit outside the chamber, and (3) one person activating water-deluge fire suppression system	Designation of special job function enhances safety in case of emergency

Reference: Swanson et al., 1999; Kot, 2005.

can be done in a multiplace chamber provided that several conditions are met (Table 18-9).

✔ Most *implanted* cardiac pacemakers can be used safely in the hyperbaric chamber for pressures below 3 atmospheres.

Most implanted cardiac pacemakers can be used safely in the hyperbaric chamber for pressures below 3 atmospheres (Lafay et al., 2008). It is highly recommended that continuous monitoring of the ECG pattern and a defibrillator be available during every hyperbaric procedure. For patients who require temporary external pacemakers, any untested units should keep the pacemaker unit outside the chamber by using wires through the chamber wall (Kot, 2005).

MECHANICAL VENTILATION IN HYPOBARIC CONDITION

✔ Transfer of patients to a centralized location necessitates the use of aeromedical evacuation of the wounded, including those who require mechanical ventilation.

Conflicts between countries spread the battlefield across the globe. In terms of caring for the critically ill patients, it has become inefficient to provide comprehensive critical and surgical care to the wounded at all locations. The current trend is to stabilize the wounded and evacuate them to a centralized location for comprehensive care and rehabilitation. This strategy necessitates the use of aeromedical evacuation of the wounded, including those who require mechanical ventilation (Grissom et al., 1997). This section reviews the unique characteristics of mechanical ventilation in a hypobaric environment.

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High-Altitude Cerebral and Pulmonary Edema

✓ At 8,000 ft above sea level, the barometric pressure (P_B) is 564 mm Hg, and the calculated P_AO_2 at this altitude is only 59 mm Hg.

✓ Acute mountain sickness may include presence of headache and at least one of the following symptoms: gastrointestinal disturbances (anorexia, nausea, or vomiting), insomnia, dizziness, and fatigue.

At 8,000 ft above sea level, the barometric pressure (P_B) is 564 mm Hg, and the calculated P_AO_2 at this altitude is only 59 mm Hg (Chang, 2011). An acute drop in the P_B and P_AO_2 are the primary reasons for high-altitude illness experienced by unacclimatized persons. Acute mountain sickness includes a number of nonspecific subjective symptoms. They may include presence of headache and at least one of the following symptoms: gastrointestinal disturbances (anorexia, nausea, or vomiting), insomnia, dizziness, and fatigue. In severe high-altitude illness, cerebral edema or pulmonary edema could develop and these conditions are potentially fatal (Hackett et al., 2001).

✓ The treatment for acute mountain sickness or high-altitude cerebral edema includes one or a combination of the following: descent to a lower altitude, and use of supplemental oxygen.

High-altitude cerebral edema is related to vasodilatation of cerebral vessels, overperfusion, and inadequate volume buffering by cerebrospinal fluid. The treatment for acute mountain sickness or high-altitude cerebral edema includes one or a combination of the following: descent to a lower altitude, and use of supplemental oxygen. When descent is not possible or supplemental oxygen is not available, acetazolamide (Diamox) and dexamethasone may be useful to treat the symptoms associated with acute mountain sickness. In the event that high-altitude cerebral edema does not respond to the initial treatments, further descent or use of a portable hyperbaric chamber may be necessary (Hackett et al., 2001).

✓ High-altitude pulmonary edema is noncardiogenic, but it is associated with pulmonary hypertension and elevated capillary pressure.

High-altitude pulmonary edema is primarily noncardiogenic in origin, but it is associated with pulmonary hypertension and elevated capillary pressure. The signs and symptoms of this type of pulmonary edema may include decreased endurance, dry cough, pink or bloody sputum, resting tachycardia, and tachypnea. Chest radiography and breath sounds resemble typical pulmonary edema (Hultgren, 1996).

✓ The treatments for high-altitude pulmonary edema include descent to lower altitude, supplemental oxygen, and a portable hyperbaric chamber.

High-altitude pulmonary edema accounts for most fatalities from high-altitude illness. As is the case for acute mountain sickness, the incidence of high-altitude pulmonary edema is related to the rate of ascent to the high altitude, the altitude reached, individual susceptibility, and exertion. Cold can be a contributing factor as it causes sympathetic stimulation and a resulting increase in pulmonary artery pressure (Reeves et al., 1993). The treatments for high-altitude pulmonary edema include descent to lower altitude, supplemental oxygen, and use of a portable hyperbaric chamber. Nifedipine, a calcium channel blocker, may be used when descent or medical equipment and supplies are not available. Nifedipine relieves acute pulmonary edema due to its antihypertensive property, which effectively reduces the pulmonary artery pressure (Oelz et al., 1989).

Airplane Cabin Pressure

✓ For the safety and comfort of the passengers inside the commercial airplanes, most airplanes are pressurized to a cabin pressure altitude of 8,000 ft (range from 5,000 ft to 8,000 ft).

Most commercial airplanes travel at a cruising altitude between 25,000 ft and 40,000 ft. At high altitudes, the air becomes thinner (less dense) and this condition lowers the airflow resistance to the airplane. Since less thrust is needed to move at a given speed, the airplanes can fly more efficiently at higher altitudes. A lower cabin pressure also makes the airplane lighter and consumes less fuel. For the safety and comfort of the passengers inside the airplane, most airplanes are pressurized to a cabin pressure altitude of 8,000 ft (range from 5,000 ft to 8,000 ft).

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TABLE 18-10 Tolerated SpO_2 at Different Cabin Pressure Altitudes

Altitude (MSL)	Tolerated SpO_2
Sea level	95% to 100%
10,000 ft*	88% to 93%
13,000 ft	83% to 88%
16,000 ft	75% to 80%
20,000 ft	70% to 75%

**Commercial airplane cabin pressure altitude ≤ 8,000 ft.*

✓ At an altitude of 8,000 ft, the low P_AO_2 (about 59 mm Hg) leads to high-altitude hypoxia.

At an altitude of 8,000 ft, the low P_AO_2 (about 59 mm Hg) leads to high-altitude hypoxia. In turn, hypoxia and related physiologic changes necessitate the use of supplemental oxygen (Hackett et al., 2001). Federal Aviation Administration (FAA) regulations in the U.S. mandate that the cabin pressure altitude may not exceed 8,000 ft at the maximum operating altitude of the airplane under normal operating conditions (Federal Aviation Regulation, 1996). The FAA also requires supplemental oxygen for pilots operating at cabin pressure altitudes above 12,500 ft for over 30 minutes, and at all times over 14,500 ft mean sea level (MSL).

hypobaric condition: an environment in which the atmospheric pressure is less than one barometric pressure (e.g., high altitude, airplane cabin in flight).

During flight under **hypobaric condition**, the *tolerated* oxygen saturation can be lower than the normal oxygen saturation for home altitude. At 10,000 ft cabin pressure altitude MSL, the tolerated oxygen saturation ranges from 88% to 92%. As a general guideline, supplemental oxygen should be used when a person's pulse oximetry measurement is 10% below the normal value for home altitude (avweb.com, 1999). Table 18-10 shows the tolerated SpO_2 at different altitudes (MSL).

✓ As a general guideline, supplemental oxygen should be used when a person's pulse oximetry measurement is 10% below the normal value for home altitude.

Ventilator Parameter Changes under Hypobaric Conditions

✓ During mechanical ventilation at high altitudes, the measured tidal volume and peak flow increase as the barometric pressure decreases.

For mechanically ventilated patients, traveling at high altitudes presents technical and physiological challenges. Technically, the tidal volume, peak flow, and minute ventilation show moderate changes from the measurements that are expected at sea level (Schedler et al., 2007; Thomas et al., 1994). Table 18-11 shows the changes in volume and flow measurements under hypobaric conditions with P_B ranging from 695 mm Hg to 524 mm Hg. The measured tidal volume and peak flow increase as the barometric pressures decreases (Schedler et al., 2007).

Another important finding in Table 18-11 is the inaccurately indicated V_T at all P_B levels. At an altitude of 2,500 ft, the indicated V_T shows 371 mL (with a set V_T of 500 mL). This discrepancy in tidal volumes could cause confusion in the mind of the operator as to the proper functioning of the ventilator.

✓ During mechanical ventilation on ascent to a high altitude, the continuing increase in delivered tidal volume (result of gas expansion during ascent) can cause hyperinflation and become potentially harmful to the patient.

Physiologically, a spontaneously breathing patient may suffer hypoxia due to the reduced P_AO_2 at high altitudes. During mechanical ventilation on ascent to a high

TABLE 18-11 Changes in Ventilator Parameters under Hypobaric Conditions

Altitude in ft	**2,500**	**5,000**	**8,500**	**10,000**
P_B in mm Hg	695	633	554	524
Set V_T in mL (Evita 4 Drager)	500	500	500	500
Indicated V_T in mL (Evita 4 Drager)	371	355	307	353
Measured V_T in mL (ASL 5000 lung simulator, Ingmar Medical)	512	521	567	647
Peak Flow in mL/s	1158	1197	1337	1554

(Data rounded to whole numbers and ranges not included. Reference: Schedler et al., 2007.)

altitude, the continuing increase in delivered tidal volume (result of gas expansion during ascent) can cause hyperinflation and become potentially harmful to the patient. The increase in tidal volume, airway, and alveolar pressures may produce lung injuries or volutrauma if the condition is unrecognized. (Abadia de Barbara, 2004).

✓ Pressure-compensated ventilators tend to deliver stable tidal volume, peak inspiratory flow, peak proximal airway pressure, and minute ventilation.

Pressure Compensation

Since most airplanes cruise at a cabin pressure altitude of up to 8,000 ft, the tidal volume of non-pressure-compensated ventilators should be monitored and adjusted during airplane ascent and descent.

Pressure-compensated ventilators are better suited for mechanically ventilated patients who must travel long distance at high altitude. Pressure-compensated ventilators tend to deliver stable tidal volume, peak inspiratory flow, peak proximal airway pressure, and minute ventilation (Grissom et al., 1997).

✓ For ventilators without the capability of **pressure compensation,** they may be recalibrated after significant changes in cabin altitude.

pressure compensation: a ventilator feature that makes self-adjustment of pressure or volume output based on changing atmospheric pressure.

Use of Non-Pressure-Compensated Ventilators. For ventilators without the capability of **pressure compensation**, they may be recalibrated after significant changes in cabin altitude. Manual ventilation is often necessary to maintain adequate ventilation and oxygenation for the patient during the calibration period (Grissom et al., 1997). Another method of compensation is to monitor the ventilator outputs (tidal volume, frequency, and minute ventilation) and make necessary adjustments to the tidal volume and frequency during ascent and descent. Methods of adjustment will be discussed under *Adjustment of Tidal Volume* in this chapter.

TRAVELING WITH PORTABLE VENTILATORS

Improved technology and miniaturization of ventilator components enable more ventilator-dependent patients to travel by car or air. The major drawback of traveling by air is the lack of flexibility. A patient must go by the time schedules determined

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and managed by other agencies. Other traveling issues include movement and storage of medical devices (e.g., wheelchair, ventilator, backup batteries, oxygen, suctioning devices), personal traveling companion, availability of electricity in flight, and a backup ventilator at the destination. This section reviews the issues related to portable ventilators while traveling by air.

Characteristics of Portable Ventilators

✔ Capability, battery life, weight, dimensions, and alarms of the portable ventilator are crucial factors in making a travel plan.

There are many portable ventilators capable of providing mechanical ventilation away from home. Obviously, the most important feature is that the portable ventilator can provide the modes and settings to suit the patient's needs. Battery life, weight, dimensions, and alarms of the portable ventilator are other crucial factors in making a travel plan. Table 18-12 shows the features of four portable ventilators.

Traveling in the United States

Prior to making travel plans, the traveler should obtain from the physician a permission verifying that the person is fit to travel based on the current medical condition. Travel arrangements should also be made with the medical care team and home care agency at the destination in the event that such services become necessary. Extra supplies should be available in flight.

✔ It is important to emphasize to the airline contact persons that a portable ventilator is different from portable oxygen.

Advance planning is a necessity for using a ventilator in flight. The initial contacts with the commercial airline typically involve the reservation agent and customer representative. It is important to emphasize to these contact persons that a portable ventilator is different from portable oxygen. The person making the flight reservation should be able to provide the medical "approval to travel," detailed technical specifications of the ventilator, and the contact information of the ventilator manufacturer. The airline engineering and medical departments may need this information for the approval process.

✔ The FAA does not require the airline to provide electrical connection for the portable ventilator. The traveler should bring extra battery packs.

Use of the airplane's electrical connection is at the discretion of the airline, since the FAA does not require the airline to provide this crucial service. The ventilator and extra battery packs must fit under the seat. If the ventilator does not fit under the seat, the ventilator user may hold the ventilator or purchase another seat. The traveler should be able to maneuver about the aircraft with minimal assistance from the flight attendant or passenger assistant. A personal traveling companion may be required if the traveler needs a higher level of service (e.g., is wheelchair-dependent) and medical support (e.g., needs IV medication or fluid administration, endotracheal suctioning).

For users who do not require continuous ventilation (e.g., nocturnal use of BiPAP for obstructive sleep apnea), the noninvasive ventilator may be packed as a carry-on item. It should not be checked in as a luggage due to the delicate nature of the ventilator.

The FAA requires all airlines to provide a Complaints Resolution Officer at all times to resolve any disagreements between the carrier and passengers. For complete rules and regulations, visit the Accessible Journeys (www.accessiblejourneys.com) or

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TABLE 18-12 Selected Portable Ventilators Suitable for Traveling

Ventilator	Mode	Settings	Battery Weight/Dimension	Alarms
LTV 800 (Carefusion) *Pediatric use (>5 kg)*	Spontaneous Control A/C SIMV	V_T (50–2000 mL) f (10–100/min) PEEP (0–20 cm H_2O)	Internal (1 hr) External (3, 4, 9 hrs) Car cigarette lighter adaptor Weight (12.9 lbs) 3"H × 10"W × 12"D	High/low pressure Low/depleted battery Low min vent Apnea Power failure Malfunction Disconnect
UniVent Eagle 754 (Impact Instrumentation, Inc.)	A/C SIMV CPAP	V_T (0–3000 mL) f (1–150/min) PEEP (1–20 cm H_2O)	Internal (3 hrs) External (11–15 V) Weight (13 lbs) 8.87"H × 11.5"W × 4.5"D	High/low pressure Low battery V_T Power failure Malfunction Disconnect
V-Leonardo (Dima Italia S.r.l.)	Assist Control A/C SIMV	V_T (100–1600 mL) f (5–99/min) PEEP (0–15 cm H_2O)	Internal (10 hrs) External (12 VDC) Weight (20.8 lbs) 9.06"H × 14.96"W × 13"D	High/low insp pressure High exp pressure Apnea Low battery Power failure Malfunction
LP-10 (Puritan Bennett) *Discontinued in November 2006 (some remain in use)*	A/C SIMV Pressure cycle	V_T (100–2000 mL) f (1–20/min or 22–38/min) PEEP (n/a)	Internal (1 hr) External deep cycle (10–20 hrs) Weight (35 lbs) 9.75"H × 14.5"W × 13.25"D	High/low pressure Power failure Malfunction

Reference: IVUN (2010).

FAA (http://www.faa.gov/) websites to review the *Air Carrier Access Act* for people with disabilities (Accessible Journeys, 2010; IVUN, 2010).

Adjustment of Tidal Volume

When an airplane takes off and ascends to a higher altitude, a lower barometric pressure causes the gas density to decrease. A lower gas density allows the

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gas volume to increase. For users of a non-pressure-compensated ventilator while traveling by air, the set tidal volume should be decreased during ascent to prevent hyperinflation.

Conversely, during landing of an airplane, an increasing barometric pressure causes the gas density to increase. A higher gas density compresses the gas volume. This means the delivered volume decreases during landing. To compensate for loss of volume, the set tidal volume should be increased during descent.

✓ One study estimates that the tidal volume can be *reduced* by 3% per 1,000 ft of *ascent* (or *increased* by 3% per 1,000 ft of *descent*).

One study estimates that the tidal volume can be *reduced* by 3% per 1,000 ft of *ascent* or *increased* by 3% per 1,000 ft of *descent* (Schedler et al., 2007). Another method for volume compensation is to monitor the ventilator outputs (tidal volume, frequency, and minute ventilation) and make adjustments to the tidal volume and frequency during ascent and descent.

Portable Oxygen Concentrator

Most commercial airplanes use 8,000 ft of cabin pressure altitude at any cruising altitude or speed. At this cabin pressure, it is equivalent to a barometric pressure of 564 mm Hg or a P_AO_2 of 59 mm Hg. Since arterial oxygen saturation is in part determined by the gas tension, a reduced P_AO_2 can lead to hypoxia and associated physiologic changes (e.g., hyperventilation, tachycardia, tachypnea).

Altitude hypoxia affects all individuals, but it exerts a more profound influence on those with preexisting heart or lung diseases. Hypoxia induced by high altitudes can be treated with supplemental oxygen. For intermittent users of ventilators, oxygen therapy is recommended while breathing spontaneously. Prophylactic oxygen therapy reduces the work of breathing and alleviates the development of ventilatory failure due to prolonged exacerbation (Luks et al., 2007).

✓ The Air Carrier Access Act does not require airlines to provide oxygen during flights.

The Air Carrier Access Act does not require airlines to provide oxygen during flights. Some airlines charge a fee for providing supplemental oxygen per flight segment. Airlines may allow passengers to bring a portable oxygen concentrator (POC) on board. Again, as with a portable ventilator, planning must be made ahead of time to ensure smooth and safe travel. In 2005, the FAA approved several POCs for air travel. Examples of the approved POCs that weigh less than 10 lbs, along with their maximum battery duration, include: AirSep FreeStyle (4.4 lbs, 10 hrs), Delphi Central Air (9.8 lbs, 3 hrs 25 min), Inogen One G2 (7 lbs, 8 hrs), Invacare XPO2 (6 lbs, 7 hrs), Inova Labs LifeChoice (4.9 lbs, 5 hrs), and Phillips Respironics EverGo (8.5 lbs, 12 hrs).

✓ The rule of thumb is to have enough battery life to power the POC for at least 150% of the one-way travel time.

Since airlines are not required to provide direct-current electricity for the POC, a passenger must bring along sufficient batteries to power the POC. The rule of thumb is to have enough battery life to power the POC for at least 150% of the one-way travel time. The POC and batteries must be able to fit under the seat or on the lap. Refer to the Federal Aviation Administration website (www.faa.gov) for current rules and regulations on air travel with a POC or portable ventilator.

SUMMARY

Mechanical ventilation in nontraditional settings differs from that delivered in the hospital. Outside the hospital, one major difference is the lack of an array of monitors, specialized equipment, and medical support personnel. Providers of mechanical ventilation in nontraditional settings often work alone or with a small team. They must be knowledgeable, adaptive, and able to make appropriate ventilator adjustments based on the unusual or changing environment.

Self-Assessment Questions

1. Dr. Kingston asks the therapist to evaluate a patient in the long-term care unit for continuation of ventilator care in the home. The therapist should evaluate all of the following *except*:

   A. age of patient.
   B. clinical stability of the patient.
   C. type of ventilator suitable for the patient.
   D. laboratory and clinical assessment of the patient.

2. A patient with COPD has an admitting diagnosis of lobar pneumonia affecting three of five lobes. When this pulmonary problem triggers a rapid deterioration of the patient's clinical status, it is called:

   A. acute respiratory acidosis.
   B. acute exacerbation of COPD.
   C. chronic ventilatory failure.
   D. combined acidosis.

3. Which of the following arterial blood gas (ABG) reports reflects a condition of exacerbation of COPD?

   A. pH = 7.26, $PaCO_2$ = 78 mm Hg, PaO_2 = 44 mm Hg, HCO_3^- = 34 mEq/L
   B. pH = 7.40, $PaCO_2$ = 45 mm Hg, PaO_2 = 81 mm Hg, HCO_3^- = 27 mEq/L
   C. pH = 7.38, $PaCO_2$ = 54 mm Hg, PaO_2 = 56 mm Hg, HCO_3^- = 31 mEq/L
   D. pH = 7.49, $PaCO_2$ = 30 mm Hg, PaO_2 = 43 mm Hg, HCO_3^- = 22 mEq/L

4. Ms. Lange has been receiving around-the-clock mechanical ventilation for two months due to a head injury sustained in an automobile accident. Prior to transfer to her home for ventilator care, which of the following is the most essential requirement, based on her condition?

   A. high and low pressure alarms
   B. live-in health care assistance
   C. backup ventilator
   D. insurance coverage

5. In a mass casualty incident, START or JumpStart is done to:

   A. recharge the crash cart battery.
   B. calculate the joules level for defibrillation.
   C. categorize patients based on medical condition.
   D. speed up the rescue efforts.

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6. The Simple Triage and Rapid Treatment algorithm uses which of the following three physiologic parameters?

   A. Pulse oximetry, Pulse, Blood pressure
   B. Respiration, Perfusion, Mental status
   C. Respiration, Perfusion, Blood pressure
   D. Pulse oximetry, Mental status, Blood pressure

7. Which of the following triage systems incorporates life-saving intervention (LSI) in its algorithm?

   A. START
   B. SOFA
   C. JumpSTART
   D. SALT

8. The sequential organ failure assessment (SOFA) score is done to:

   A. evaluate the functions of major organs.
   B. determine the treatments required for critically ill patients.
   C. predict the outcomes of critically ill patients.
   D. obtain biopsies from major organs.

9. The estimated number of portable ventilators in the U.S. Strategic National Stockpile program is about:

   A. 500.
   B. 5,000.
   C. 10,000.
   D. 100,000.

10. Normal metabolism in cells and tissues require a minimum of _______ of oxygen and this amount can be made available by using _______ of F_IO_2 at _______ atmospheric pressures.

    A. 2 vol%; 21%; 1
    B. 2 vol%; 21%; 3
    C. 6 vol%; 100%; 1
    D. 6 vol%; 100%; 3

11. Indications for a hyperbaric chamber include all of the following conditions *except*:

    A. severe carbon dioxide necrosis.
    B. severe carbon monoxide poisoning.
    C. diver's decompression sickness.
    D. gas gangrene.

12. An airplane is cruising at an altitude of 35,000 ft and its cabin is pressurized to an altitude of 8,000 ft. The calculated P_AO_2 in the cabin is about:

    A. 59 mm Hg.
    B. 80 mm Hg.
    C. 150 mm Hg.
    D. 600 mm Hg.

13. Travelling at a high altitude on a commercial airplane, supplemental oxygen is recommended when a person's SpO_2:

    A. drops to 90%.
    B. is 10% below the normal value for home altitude.
    C. is lower than 95%.
    D. is 5% below the normal value for home altitude.

14. To compensate for the changes in tidal volume during airplane takeoff, study has indicated that the tidal volume can be _______ by _______ per 1,000 ft of *ascent*.

    A. increased; 3%
    B. increased; 10%
    C. decreased; 3%
    D. decreased; 10%

15. High-altitude-induced hypoxia may cause all of the following initial physiologic changes *except*:

    A. hyperventilation.
    B. tachypnea.
    C. tachycardia.
    D. hypoventilation.

Answers to Self-Assessment Questions

1. A.
2. B.
3. A.
4. C.
5. C.
6. B.
7. D.
8. C.
9. C.
10. D.
11. A.
12. A.
13. B.
14. C.
15. D.

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Additional Resources

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Chapter 19

Case Studies

Case 1: **COPD**
Wayne Lawson

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Case 4: **Head Injury**
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Michell Oki

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Paul G. Eberle

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Case 11: **Guillain-Barré**
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Case 12: **Botulism**
Paul G. Eberle

Case 13: **Meconium Aspiration/Patent Ductus Arteriosus**
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Case 14: **Persistent Pulmonary Hypertension of the Newborn**
Lisa M. Trujillo

Case 15: **Home Care and Disease Management**
Angie Roberts

Case 16: **End-of-Life Sedation on Mechanical Ventilation**
Jonathan Waugh

NOTE: Blood gas values for cases 2 to14 were measured and reported at P_B 640 mm Hg (4,400 ft elevation) near Salt Lake City, Utah.

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CASE 1: COPD

INTRODUCTION

✔ The inability to breathe comfortably unless in an upright position is called orthopnea and is a common clinical sign of pulmonary artery congestion or congestive heart failure.

✔ Sudden weight gain with dependent edema in a COPD patient is a sign of cor pulmonale.

A 74-year-old Caucasian male with a diagnosis of pulmonary emphysema, made six years prior, was seen in the emergency department with a complaint of shortness of breath. He has had respiratory problems on and off since diagnosis, including two hospital admissions, each of several days duration. He stated he caught a cold the previous week that moved down into his chest, and since that time breathing has become increasingly more difficult. He related that in his usual state of health, he was able to move freely about his home and yard and enjoyed his hobby of gardening, but now was unable to do either. Sleeping in bed had become such a problem that for the previous two nights he slept sitting back in his easy chair. His normal sputum production of about a tablespoon per day had increased to about 1/4 cup a day and had turned from white to yellow in color. He gained 6 lb in the past 4 days and noticed that his ankles were swollen by the end of the day. When questioned about his smoking history, he stated that he had smoked two packs per day for 40 years, and had tried unsuccessfully to quit after his diagnosis of emphysema was made. He now smokes a half pack per day of a "lighter" brand. His home medications include an albuterol metered-dose inhaler (MDI), 1 to 2 puffs every 2 to 6 hours, as needed.

Physical Examination

General. The patient is a mildly obese male, weight 100 kg, height 72 in., in moderately severe respiratory distress, sitting on the edge of the bed leaning forward supporting his weight with his palms and breathing through pursed lips.

Vital Signs. Heart rate 124/min, blood pressure 150/90 mm Hg, frequency 28/min, and temperature 100.5°F.

HEENT. Some cyanosis of the lips, otherwise unremarkable.

Neck. Trachea in the midline, no masses, stridor, lymphadenopathy, or thyromegaly. Carotid pulses ++ without bruit. There is marked use of accessory muscles of the neck with mild jugular venous distention.

✔ Paradoxical motion of the abdomen during ventilation is an indication of diaphragmatic muscle fatigue.

Chest. The anteroposterior diameter of the chest is increased with a deep suprasternal notch and some paradoxical motion of the abdomen. Decreased tactile fremitus and absent point of maximal impulse (PMI) are noted with hyper-resonance to percussion bilaterally.

Heart. Sounds are distant with no irregularity in rate or rhythm noted; no gallops or murmurs.

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Lungs. Bilaterally diminished with scattered expiratory wheezing, bibasilar rhonchi, and a prolonged expiratory phase.

Abdomen. Mild hepatomegaly, paradoxical movement with breathing, otherwise unremarkable.

Extremities. Slight digital cyanosis with +2 pitting edema in both ankles.

Initial Assessment and Treatment

In the emergency room, a portable chest radiograph and arterial blood gas with co-oximetry were obtained, the patient was placed on a 2 L/min of oxygen via nasal cannula and given an aerosol treatment with 2.5 mg albuterol sulfate in NS. Room air blood gas results revealed:

pH	7.32
$PaCO_2$	70 mm Hg
PaO_2	44 mm Hg
HCO_3^-	35 mEq/L
BE	+6
SaO_2	80%
Hb	16 g/dL
HBCO	3%

✓ Acute-on-chronic ventilatory failure in the COPD patient requires immediate medical attention.

✓ Aminophylline (theophylline) may help to improve diaphragmatic function in the COPD patient.

✓ Corticosteroids are only useful in the treatment of COPD if the patient has evidence of reversible airway disease.

The chest radiograph revealed evidence of hyperinflation, an increase in vascular markings, and infiltrates in the RLL. Because of the patient's clinical condition, chest radiograph findings, and the abnormal blood gases (acute ventilator failure superimposed on chronic ventilatory failure), the patient was transferred immediately to the medical intensive care unit (ICU) for further evaluation and treatment. The patient was then started on the following regimen: supplemental oxygen at 2 L/min by nasal cannula, nebulized albuterol sulfate, 2.5 mg in NS Q2H, 2.5 mg/kg loading dose of aminophylline over 30 min, followed by a maintenance dose of 0.5 mg/kg per hour IV (titrated according to serum levels), furosemide, 40 mg, IV push, and methylprednisolone, 120 mg IV, Q6H. Sputum for Gram stain and culture was obtained and sent to the lab. The patient was also started on a prophylactic broad-spectrum antibiotic. The report on the Gram stain came back later and showed numerous gram-positive diplococci.

Indications

Over the next hour, the patient's respiratory status continued to deteriorate despite intervention. Respiratory frequency rose to 36/min and paradoxical abdominal motion became more pronounced. The rising frequency in conjunction with the abdominal paradox and the increasing $PaCO_2$ from the first blood gas are indicative of increasing ventilatory fatigue. Impending acute ventilatory failure must be assumed.

Initial Settings

On the basis of clinical and laboratory data, a decision was made to assist the patient's ventilation. In lieu of endotracheal intubation, bilevel positive airway pressure (bilevel PAP or BiPAP®) was initiated via nasal mask at the settings below. The IPAP and EPAP levels were titrated to 15/5 cm H_2O resulting in an SpO_2 of around 90%.

✓ In the alert and cooperative patient with intact airway defenses, noninvasive ventilation can provide ventilatory support without the risks associated with intubation.

Mode	Spontaneous
IPAP	15 cm H_2O
EPAP	5 cm H_2O
Sup. O_2	2 L/min

The patient was poorly compliant with the therapy, removing the mask frequently, complaining of "not getting enough air." After one hour, an arterial blood gas was obtained and revealed the following:

pH	7.25
$PaCO_2$	80 mm Hg
PaO_2	56 mm Hg
HCO_3^-	34 mEq/L
BE	+3
SaO_2	86%
Hb	16 g/dL
HBCO	2%

On the basis of the worsening ventilatory failure despite noninvasive ventilatory assistance, the patient was intubated with a size 8.5 endotracheal tube, and volume-controlled ventilation was initiated on the following settings:

Mode	CMV
V_T	750 mL
f	10/min
Flow	55 L/min
F_IO_2	0.40
PEEP	5 cm H_2O
Flow Trigger	3 L/min

After 30 min, an arterial blood gas was obtained and revealed the following:

pH	7.37
$PaCO_2$	65 mm Hg
PaO_2	88 mm Hg
HCO_3^-	36 mEq/L
BE	+6
SaO_2	96%
Hb	+6 g/dL
HBCO	1%

On the basis of the blood gas, an order was written to titrate the patient's F_IO_2 to maintain the $SpO_2 \geq 92\%$. No other changes were made to the ventilatory parameters. Albuterol orders were changed to MDI, 6 puffs inline Q4H. The patient was suctioned prn for moderate amounts of thick pale yellow secretions.

Patient Monitoring

✓ In order to minimize theophylline toxicity, the therapeutic range for serum theophylline level should be kept from 5 to 15 mcg/mL.

Over the course of the next 72 hours the patient was rested on the ventilator and treated appropriately for his pneumonia and right heart failure. The patient remained alert and cooperative with his care. A chest radiograph done on day three of ICU admission demonstrated clearing of the pneumonic process in the RLL. Findings characteristic of emphysema were also present, including hyperlucent lung fields, a flattened diaphragm, widely spaced ribs, and a narrow heart shadow. Serum theophylline levels were monitored daily, averaging 9 mcg/mL.

The ventilator settings were adjusted appropriately and currently are:

Mode	SIMV
V_T	750 mL
f	6/min
PIF	55 L/min
F_IO_2	0.35
PEEP	5 cm H_2O
PS	7 cm H_2O
Flow Trigger	3 L/min

Spontaneous Parameters:

Spont. f	12/min
Spont. V_T	550 mL
VC	2.21 L
MIP	−36 cm H_2O

The arterial blood gas drawn on the current ventilator settings shows:

pH	7.39
$PaCO_2$	58 mm Hg
PaO_2	74 mm Hg
HCO_3^-	34 mEq/L
BE	+7
SaO_2	94%
Hb	15.5 g/dL
HBCO	1%

Patient Management

The patient's vital signs have normalized and, along with ventilator care, fluid status was normalized and bronchodilator therapy was continued to relieve bronchospasm and help promote mucociliary clearance. Antibiotic therapy was continued and adjusted on the basis of the culture and sensitivity report. Secretion volume and consistency have decreased and color has changed from yellow to white.

Weaning

As the patient's condition continued to improve, ventilatory support was gradually withdrawn over the next 3 days. He was placed on 5 cm H_2O of CPAP at an F_IO_2 of 0.35 with pressure support of 5 cm H_2O. After 4 hours, spontaneous ventilatory parameters were measured and an arterial blood gas was obtained. The results are as follows:

f	18/min
V_T	525 mL
V_E	9.45 L
f/V_T	34/min/L
VC	2.85 L
MIP	−44 cm H_2O
pH	7.38
$PaCO_2$	59 mm Hg
PaO_2	68 mm Hg
HCO_3^-	34 mEq/L
BE	+7
SaO_2	92%
Hb	15.1 g/dL
HBCO	1%

f/V_T ratio <100/min/L is predictive of weaning success.

Complications

On the basis of the patient's clinical condition and diagnostic results, he was extubated and placed on 2 L/min O_2 via nasal cannula. He was moved to the medical floor later that day, and was subsequently discharged 2 days later after being enrolled in the hospital's comprehensive outpatient rehabilitation program. His home medications included Combivent (albuterol and ipratropium) via MDI and oral theophylline.

A comprehensive rehabilitation program teaches the patient about the disease process, self-care, diet, maintenance of an active lifestyle, and smoking cessation.

CASE 2: STATUS ASTHMATICUS

INTRODUCTION

P.W., a 32-year-old, 48-Kg female, was gravida 14, para 0-4-9-4, and about 21½ weeks pregnant at the time of her admission. She was evaluated for acute exacerbation of asthma with a concomitant upper respiratory tract infection. The patient stated that she was "doing fine" until approximately midnight at which time she began having difficulty breathing. Her dyspnea continued despite taking her routine home-inhaled bronchodilators.

Aminophylline is commonly used to reverse severe bronchospasm in acute asthma that has not responded to epinephrine.

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Hypoxemia is severe since the PaO_2 is only 59 mm Hg with a non-rebreather mask.

In the emergency room, she was tachypneic with inspiratory and expiratory wheezes, had diffuse rhonchi, and dry crackles throughout all lung fields. She was placed on oxygen with a non-rebreather mask at 10 L/min and given one Proventil® nebulizer treatment. A stat blood gas was done and she was started on IV aminophylline. Her respiratory frequency at that time was 28/min, with the following blood gas results: *(P_B of 640 mm Hg at 4,400 ft elevation maintains a normal PaO_2 of approx. 70 mm Hg).*

$PaCO_2$ of 34 mm Hg indicates hyperventilation is needed to maintain borderline oxygenation.

pH	7.48
$PaCO_2$	34 mm Hg
PaO_2	59 mm Hg
HCO_3^-	24.2 mEq/L
Hb	12.8 g %
SpO_2	91%
Mode	Non-rebreather mask
Oxygen	10 L/min
f	Spontaneous 28/min

Increase in frequency from 28 to 36/minute is an ominous sign in the progression of acute asthma.

Four hours later her frequency had increased to 36/minute and she had received medication nebulizer treatments every hour with Proventil®. However, inspiratory and expiratory wheezes persisted. A follow-up blood gas revealed:

PaO_2 does not show much improvement in spite of increase in spontaneous frequency.

pH	7.43
$PaCO_2$	36 mm Hg
PaO_2	58 mm Hg
HCO_3^-	23.2 mEq/L
Hb	12.8 g %
SpO_2	91%
Mode	Non-rebreather mask
Oxygen	10 L/min
f	Spontaneous 36/min

Indications

Subsequent blood gases indicated that the patient's ventilatory status quickly deteriorated and she became unable to maintain prolonged hyperventilation. Her $PaCO_2$ began rising toward normal (35 to 45 mm Hg).

The $PaCO_2$ rising from a low level (by hyperventilation) toward normal is indicative of muscle fatigue. Impending ventilatory failure is likely if not treated aggressively.

In asthma, the increased work of breathing is related to a combination of bronchospasm, airway inflammation, and mucus accumulation in the airway. The changes in the airway increase the airflow obstruction, airway resistance, and work of breathing. They also perpetuate degranulation of the mast cells causing release of histamine and other harmful chemical substances. Asthmatic patients may not respond to inhaled bronchodilators and eventually may show signs of exhaustion with hypersomnolence, progressive hypoxemia, and ultimately hypercapnea.

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Based on the clinical signs and anticipated progression of asthma, the decision was made to perform elective intubation and prophylactic mechanical ventilation.

Initial Settings

The patient was initially set on assist/control (AC) mode at a frequency of 10/min, V_T of 500 mL (approx. 10 mL/Kg ideal body weight), F_IO_2 of 90%, and PEEP of 5 cm H_2O.

Ideal Body Weight:

Male: 50 + 2.3 (Height in Inches − 60) × 6 mL
Female: 45.5 + 2.3 (Height in Inches − 60) × 6 mL

The peak flow was increased to match her inspiratory demand, approaching 110 L/min due to her high inspiratory flow rate requirement. Every effort was made to reduce the work of breathing and decrease her anxiety level from a ventilatory standpoint. Blood gases obtained revealed the following:

pH	7.43
$PaCO_2$	33 mm Hg
PaO_2	55 mm Hg
HCO_3^-	21.3 mEq/L
Hb	12.4 g %
SpO_2	90%
Mode	A/C
f	10/min
V_T	500 mL
F_IO_2	90%
PEEP	5 cm H_2O

PC-IRV may improve oxygenation and minimize occurrence of barotrauma.

Although pharmacologic sedation is generally not recommended for asthmatic patients, she was medicated and changed to pressure-controlled inverse ratio ventilation (PC-IRV) mode at a 2:1 ratio to improve oxygenation and prevent barotrauma related to positive pressure ventilation. She was started at a frequency of 20/min, an inspiratory pressure of 40 cm H_2O, T_I of 0.5 sec, F_IO_2 of 60%, and PEEP of 10 cm H_2O. Blood gases were as follows:

pH	7.36
$PaCO_2$	44 mm Hg
PaO_2	64 mm Hg
HCO_3^-	24 mEq/L
Hb	11.8 g %
SpO_2	92%
Mode	PC-IRV
T_I	0.5 sec
I:E ratio	2:1
f	20/min
PIP	40 cm H_2O
F_IO_2	60%
PEEP	10 cm H_2O

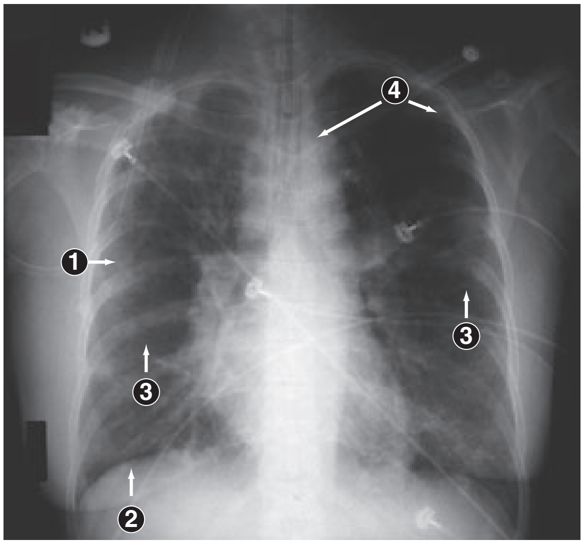

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FIGURE 19-1 Status asthmaticus. Hyperinflation is evident on this chest radiograph since the distance between the adjacent ribs in widened (1) and the diaphragm is depressed (2). Infiltrates or areas of edema are noted (3). The central venous line (4) is also visible on this chest radiograph.

No changes in her care or ventilator parameters were made at that time. Breath sounds revealed wheezes and coarse rhonchi throughout all lung fields. She continued to receive her bronchodilator therapy and was suctioned appropriately.

Patient Monitoring

The chest radiograph shows the characteristic hyperinflation of the lungs. The distance between the adjacent ribs is widened and the diaphragm is depressed. Infiltrates or areas of edema are also noted on the chest radiograph (Figure 19-1). Other than these signs, the chest radiograph is normal.

✓ The therapeutic range of serum theophylline is between 5 and 15 mcg/mL.

Over the next 3 days, the ventilator settings were weaned appropriately and the settings were pressure-controlled at 30 cm H_2O, T_I of 0.7 sec for a 2:1 ratio, F_IO_2 of 50%, and PEEP of 10 cm H_2O. She was beginning to show signs of reduced inflammation of her airway with only occasional inspiratory and expiratory wheezes. Small amounts of cloudy, clear secretions were suctioned from her endotracheal tube. Peak and trough levels of serum theophylline were drawn to monitor for possible toxicity. Blood gas results were as follows:

pH	7.43
$PaCO_2$	36 mm Hg
PaO_2	78 mm Hg
HCO_3^-	23 mEq/L
Hb	12 g %
SpO_2	93%
Mode	IR-PCV
T_I	0.7 sec
I:E ratio	2:1
f	20/min

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P_{INSP}	30 cm H_2O
F_IO_2	50%
PEEP	10 cm H_2O

She was becoming more alert and oriented to time and place and was appropriately changed to SIMV with pressure support (PS) to supplement her own efforts to breathe. She appeared calm at first but became increasingly restless and combative over the afternoon and evening. She frequently motioned for the endotracheal tube to be removed and was frustrated about her inability to talk.

Patient Management

In addition to the ventilator management strategies, adequate hydration was provided to promote mucociliary clearance and bronchopulmonary hygiene. Inhaled bronchodilators, glucocorticosteroids, and mast cell stabilizers (nonacutely) were used to bring the bronchospasm and inflammation under control.

Key Medications

The patient was treated with IV aminophylline, beta adrenergics (Proventil® and Serevent®) via MDI and given inline through the ventilator circuit every 3 to 4 hours. These served to control the symptoms associated with asthma while she was started on triacinolone acetonide (Azmacort®), a corticosteroid inhaler to control inflammation. She was also started on two puffs of cromolyn sodium (Intal®) to help prevent further degranulation of mast cell responsible for hyper-reactive airway disease and future attacks.

Intal is not given in acute asthma episodes but is used as a prophylactic measure to control airway hyperreactivity.

Weaning

The weaning process was uneventful as the patient continued to improve. She was eventually placed on CPAP without pressure support for extubation trial. Her spontaneous breathing parameters revealed the following results:

$f = 15$ $\quad \dot{V}_E = 8.1$ L $\quad$ Average $V_T = 0.54$ L $\quad$ VC = 2.19 L $\quad$ MIP = −43 cm H_2O
$f/V_T = 28$/min/L

At some point, the patient extubated herself while being restrained. The decision was made to provide nasal cannula at 6 L/min, which maintained her oxygen saturations above 90%.

An f/V_T ratio (RSBI or rapid shallow breathing index) of less than 100/min/L correlates with weaning success.

Complications

Her oxygen demands decreased over several days where she continued with her bronchodilators, was weaned to room air, and was discharged after 6 days. There were no apparent complications throughout her hospital stay and she was advised

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to seek and follow medical advice through her primary care provider. She remained on an oral steroid (Prednisone®) and an antibiotic (Erythromycin®) for an additional 10 days after discharge from the hospital.

NOTE: This case study on status asthmaticus describes the evaluative, diagnostic, and ventilatory management techniques employed at the time it was first written. For additional information on the treatment of asthma, please refer to the guidelines outlined by the National Institute of Health (1997, Oct.). *Practical guide for the diagnosis and management of asthma. NIH Publication No. 97-4053.* Additionally, asthma guidelines issued by the NHLBI's National Asthma Education and Prevention Program in 2007 should be consulted. It has also been shown that people who are allergic to certain foods are *also* likely to have asthma. Information can be obtained from the National Institute of Allergy and Infectious Diseases; Dec. 2010, *126*(6), p. s1–58.

CASE 3: POST-ABDOMINAL SURGERY

INTRODUCTION

Leukocytosis (↑WBC) is generally caused by infection and is usually transient. It may also occur after hemorrhage.

Low hematocrit leads to tissue hypoxia and it may account for her symptoms of shortness of breath, fatigue, weakness, and decreased exercise tolerance.

Diabeta (glyburide) is used to treat type 2 diabetes (condition in which the body does not use insulin normally and therefore cannot control the amount of sugar in the blood).

Thrombocytopenia (↓platelets) occurs in acute infections, anaphylactic shock, and some hemorrhagic diseases and anemias.

C.T., a 78-year-old, 90-Kg, 5'4" female, was admitted to the emergency room (ER) with complaints of the following ailments that had persisted for the past several days: abdominal pain, nausea, fatigue, weakness, shortness of breath, decreased exercise tolerance, vomiting, and black tarry stools.

C.T. had a history of steroid-dependent asthma, COPD, coronary artery disease, diabetes, and arthritis. Her medications at home included Lasix, Diabeta, nitroglycerin, aspirin, Prozac, potassium, Proventil, Intal, Azmacort, and oxygen at 2 to 3 L/min.

In the ER her abdominal exam revealed severe pain in all quadrants and her abdominal radiograph showed free air in all areas. Respiratory assessments showed severe peripheral cyanosis, spontaneous respiratory frequency 32/min, heart rate 120/min, decreased breath sounds in all lobes with fine inspiratory crackles in the lower lobes, and accessory muscle use during inspiration.

Laboratory studies were done upon admission with the following results: WBC 22.2×10^3 (*normal* 3.2 *to* 9.8×10^3), hematocrit (Hct) 25% (*female average 42%*), platelets 34.2×10^3 (*normal* 130 *to* 400×10^3). The electrocardiograph (ECG) showed sinus tachycardia with nonspecific ST and T wave changes.

Other medical history was not significant. The patient's last hospitalization was 3 years ago for asthma. Upon discharge from the hospital, her room air blood gases were pH 7.43, $PaCO_2$ 50 mm Hg, PaO_2 42 mm Hg, HCO_3^- 32 mEq/L, and SaO_2 77%. She was prescribed home oxygen therapy at that time.

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Discharge $PaCO_2$ (50 mm Hg) will serve as the baseline and target value for subsequent blood gases.

After her exam in the ER, she received two units of packed red blood cells (RBCs) and one unit of plasma to treat her low hematocrit. Due to her acute and severe condition, she was taken to the operating room for abdominal exploratory surgery.

Blood transfusion is indicated because of ↓ hematocrit and ↓ platelets.

Indications

The patient was heavily sedated during her abdominal exploratory surgery and required large amounts of IV fluids, including blood products to maintain her blood pressure during surgery and to increase her low hematocrit level. A bleeding gastric ulcer and a perforated duodenal ulcer were found. They were repaired without complications. The patient was returned to the ICU, intubated, and placed on mechanical ventilation for postoperative recovery.

Initial Settings

Normal cuff pressure is between 27 and 40 cm H_2O, lower for patients with hypotension.

The patient was intubated with a size 8.0 cuffed endotracheal tube secured at the 23 cm mark at the lips. The cuff pressure was maintained at 35 cm H_2O to prevent air leak around the cuff. The volume-controlled ventilation settings were: SIMV frequency 10/min, V_T 800 mL, F_IO_2 100%, no PEEP, peak flow 45 L/min, and sensitivity −2 cm H_2O. Her initial static compliance was 74 mL/cm H_2O. Breath sounds were equal bilaterally with fine inspiratory crackles in both lower lobes. Initial blood gases after initiation of mechanical ventilation showed:

pH	7.45
$PaCO_2$	36 mm Hg
PaO_2	102 mm Hg
SaO_2	97%
HCO_3^-	24 mEq/L
Mode	SIMV
f	10/min
V_T	800 mL
F_IO_2	100%
PEEP	0 cm H_2O
Peak Flow	45 L/min
Sensitivity	−2 cm H_2O
C_{ST}	74 mL/cm H_2O

The PaO_2 is above normal and the F_IO_2 should be reduced.

Patient Monitoring

SpO_2 (96%) correlates well with the SaO_2 (97%).

The major monitoring tools used during her recovery were pulse oximetry, end-tidal CO_2 monitor, and central venous pressure (CVP). Her SpO_2 was 96%. The $P_{ET}CO_2$ was about 33 to 35 mm Hg (*normal 2 mm Hg below $PaCO_2$*), and her CVP was consistently between 10 and 12 mm Hg (*normal 1 to 7 mm Hg*).

Patient Management

CVP is higher than normal. The systemic venous volume status should be monitored closely.

Since the patient was a chronic CO_2 retainer, the target $PaCO_2$ (50 mm Hg) should be used to manage this patient.

Adjustments were made to the ventilator settings. The patient was maintained for several hours on SIMV of 10/min, V_T 800 mL, F_IO_2 40%, peak flow 45 L/min. The patient had no spontaneous ventilation after 6 hours despite being fully alert and oriented. After the F_IO_2 was decreased to 40%, the blood gas results were:

pH	7.40
$PaCO_2$	38 mm Hg
PaO_2	65 mm Hg
SaO_2	92%
HCO_3^-	23 mEq/L
Mode	SIMV
f	10/min
V_T	800 mL
F_IO_2	40%
PEEP	0 cm H_2O
Peak Flow	45 L/min

The $PaCO_2$ can be increased (from the current 38 mm Hg to patient's target 50 mm Hg) by decreasing the SIMV frequency or changing to CPAP mode (if patient can sustain spontaneous breathing).

Fluid intake was adjusted to maintain a CVP reading <10 mm Hg. Other vital signs were within normal limits for her age and condition. Proventil and Intal treatments were started for her asthma and her static compliance remained around 70 mL/cm H_2O.

The spontaneous respiratory parameters 8 hours postsurgery showed MIP −22 cm H_2O, vital capacity 700 mL. A trial of CPAP of 5 cm H_2O at an F_IO_2 of 40% was unsuccessful as she developed periods of apnea with spontaneous tidal volumes of between 300 and 350 mL.

Preexisting asthma, post-anesthesia, and tissue hypoxia are three major factors that lead to failure of CPAP trial.

Over the next several hours, her cardiopulmonary status showed steady improvement from the anesthesia effects and the patient was again tried on CPAP. She tolerated the procedure very well this time, and her spontaneous respiratory parameters at the end of the CPAP trial were: MIP −40 cm H_2O, vital capacity 1,500 mL, spontaneous frequency 16, and average V_T of 476 mL. She was successfully extubated and placed on a 50% Venturi mask and eventually weaned to nasal cannula at 3 L/min later that evening.

Since the spontaneous respiratory frequency (f) and average V_T are 16/min and 0.476 L (476 mL), respectively, the f/V_T ratio is 34/min/L (16/0.476). An f/V_T ratio of less than 100/min/L correlates with weaning success.

Some minor complications with the patient's oxygenation and perfusion status were adequately managed using oxygen, blood, and fluids. Her recovery from the abdominal exploratory surgery was gradual but uneventful, and she was discharged from the hospital a few days later.

Complications

The chest radiograph done 16 hours postextubation revealed increasing atelectasis and pneumonia in both bases with bilateral pleural effusions. Breath sounds showed increasing crackles with occasional rhonchi and wheezing. Aerosol treatments were repeated with Proventil TID and prn. Her SpO_2 on 3 L/min of oxygen was consistently in the mid- to low-80s and she was placed back on an air-entrainment mask

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at 40% during the night. The F_IO_2 was increased to 50% at times during periods of desaturation on 40% oxygen.

Her hematocrit increased from 25% to 30% (*female average 42%*) after several transfusions of Hespan, plasma, and packed red blood cells. Her most recent vital signs before discharge were: SpO_2 90%, heart rate 95/min, blood pressure 140/90 mm Hg, and CVP 20 mm Hg (*normal 1 to 7 mm Hg*). Her ABG results on discharge were similar to the baseline values from her previous hospitalization.

CASE 4: HEAD INJURY

INTRODUCTION

T.A., a 16-year-old, 52-Kg female, was brought to the emergency department after sustaining multiple traumatic injuries from a moving vehicle crash. She was a passenger in a small car involved in a high-speed collision with a truck. It was unknown if she was restrained prior to the accident but paramedics required approximately 30 to 45 min to extract her from the vehicle. She was reportedly conscious at the scene but became combative and hysterical when she arrived at the hospital. Her blood pressure (BP) was 149/87 mm Hg, pulse 104/min, temperature 34.8°C, and she was able to move all of her extremities.

Due to the extent of her injuries, she was chemically paralyzed temporarily, orally intubated, and maintained by sedation. The routine trauma laboratory studies revealed the following results: WBC count of 2.3×10^3 (*normal* 3.2 *to* 9.8×10^3), hematocrit (HCT) 37% (*female normal 42%*), platelets of 269×10^3 (*normal* 130 *to* 400×10^3), prothrombin time (PT) of 13 sec (*normal 9 to 12 sec*), and partial thromboplastin time (PTT) of 27 sec (*normal 22 to 37 sec*). Radiographs of the abdomen and chest were unremarkable. However, due to the extent of her head injuries, she was flown by air transport to a nearby trauma center.

✓ Leukopenia (decrease in WBC) may be due to the trauma or adverse effects of drugs.

Throughout transport, she was reportedly in and out of consciousness, and upon her arrival to the trauma center her BP was 119/70 mm Hg, pulse 92/min, She had a Glasgow coma score of 3 with no spontaneous eye opening and no movement of her arms or legs. During her initial assessment, she was nonverbal and noted to have a cephalhematoma in the right frontal region with a large laceration in the occipital area of her skull. Pupils were equal, round, and reactive to light. She was in a normal sinus rhythm, had equal breath sounds, and her SpO_2 was 100% while being manually ventilated via Ambu® bag on 1.0 F_IO_2.

✓ A Glasgow coma score of 3 reflects a severe coma state.

✓ Pupils that are reactive to light mean that cerebral circulation and oxygenation are adequate.

Indications

A computerized tomography (CT) scan of her head revealed an occipital condylar fracture on the right with a small intrahemispheric subdural hematoma that did

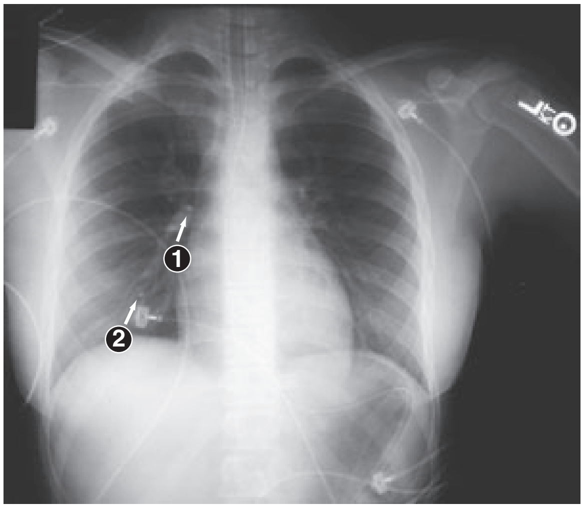

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FIGURE 19-2 Head injury. The chest radiograph is normal. The small round markings (1 and 2) are the pulmonary blood vessel running parallel to the roentgen ray (X-ray).

not require surgical intervention. Radiographs of her spine were unremarkable and there was no evidence of spleen or liver injury. The chest radiograph was also normal (Figure 19-2). She was transported to the ICU in critical condition and placed on a mechanical ventilator due to her extensive head injuries and a therapeutic drug-induced coma was initiated.

✔ A CT scan of the head and radiographs of the spine and major organs are indicated for head trauma patients.

Initial Settings

Since heavy sedation and short-term, mild hyperventilation were desired ($PaCO_2$ 34 to 35 mm Hg), the initial ventilator settings on volume-controlled ventilation consisted of assist/control (A/C) mode at a frequency of 14/min, V_T of 500 mL (approx. 10 mL/Kg), F_IO_2 of 60%, and no PEEP.

✔ In head injury, the lungs are not affected unless there are coexisting complications (e.g., flail chest, aspiration).

✔ Drug-induced coma is done to reduce metabolic rate and minimize intracranial pressure (ICP).

Patient Monitoring

The initial blood gas analysis after 20 minutes of mechanical ventilation showed:

pH	7.30
$PaCO_2$	29 mm Hg
PaO_2	256 mm Hg
HCO_3^-	13.9 mEq/L
Hb	11 gm%
Mode	A/C
f	14
V_T	500 mL
F_IO_2	60%
PEEP	0 cm H_2O

✔ An ICP of less than 10 mm Hg is the desirable target for this patient.

The patient was closely monitored. Continuous intracranial pressure (ICP) ranged from 6 to 8 mm Hg. Arterial and pulmonary artery catheters showed a heart rate of

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118/min, BP of 116/84 mm Hg, and pulmonary capillary wedge pressure (PCWP) of 11 mm Hg.

Patient Management

Mild hyperventilation is used to maintain a $PaCO_2$ between 34 and 35 mm Hg.

Carbon dioxide in the blood is a very potent vasodilator. Increased blood flow serves to promote swelling and edema following an acute injury to the brain. Blood flow to the area, and the subsequent swelling, may be reduced during the first 24 hours by maintaining the $PaCO_2$ between 34 and 35 mm Hg (can be as low as 26 mm Hg, see figure 15-3) while monitoring the ensuing edema with an ICP monitor.

Cerebrospinal fluid may also be drained to minimize the rise of intracranial pressure from subsequent swelling of the brain.

Consequently, short-term, mild hyperventilation is used as a means to reduce the $PaCO_2$ level (hypocarbia) in the blood as well as to regulate the pressure resulting from swelling of the brain after the injury. Following the insertion of the ICP monitor, the level of hyperventilation can be assessed and titrated to maintain an ICP preferentially below 10 mm Hg to minimize further injury. Judicious use of short-term hyperventilation should be monitored closely.

PEEP is contraindicated in head injuries unless severe hypoxemia is present.

The PEEP level is usually maintained at or below 5 cm H_2O because the lungs are generally not affected by head injury, and that additional pressure is transmitted to the head by the use of positive pressure to ventilate the patient. This level is desirable unless the patient's oxygen requirements exceed 60% F_IO_2, when higher levels of PEEP may be indicated.

The ventilator settings were adjusted during the next several hours and the patient was weaned to a frequency of 12/min resulting in a pH of 7.46 and $PaCO_2$ of 34 mm Hg.

Key Medications

Neurologic assessment should be done after recovery from drug-induced sedation.

The patient remained sedated with Phenytoin®, Phenobarbital®, Diazepam®, and Midazolam® during the acute phase of her hospitalization (i.e., 24 to 48 hours following the accident) to minimize the risk of further injury. After two days she was weaned to awaken for neurologic assessment.

Since the lungs were relatively unaffected, she was not given bronchodilator therapy. She was monitored for potential complications associated with artificial airway and mechanical ventilation. She was suctioned as necessary and was given periodic oral care to minimize the occurrence of infection.

Weaning

The patient was allowed to awaken fully and was changed to SIMV mode at 10/min with a pressure support level at 15 cm H_2O in an attempt to begin the weaning process. Blood gas analysis revealed the following:

pH	7.39
$PaCO_2$	40 mm Hg
PaO_2	83 mm Hg
HCO_3^-	23.3 mEq/L
Hb	13.8 g%

Mode	SIMV
f	10
V_T	500 mL
F_IO_2	60%
Pressure Support	15 cm H_2O

Weaning parameters were done each morning to include frequency, minute volume, tidal volume, vital capacity, maximum inspiratory pressure, and RSBI (f/V_T). Weaning criteria were done beginning the second day following admission ($f = 22$/min, $V_E = 9.6$ L, $V_T = 340$ mL, VC = 1.5 L, MIP = −35 cm H_2O, $f/V_T = 64$/min/L). All data suggested that her ventilatory effort was adequate and she was weaned off the ventilator. Subsequent testing showed minor neurologic deficits.

She was alert and oriented to time and place but functioned without memory of the accident. She appeared comfortable, cooperative, able to follow commands, and was not diaphoretic or febrile. She was not anxious and was breathing spontaneously on CPAP with 10 cm H_2O of pressure support at a frequency of 14/min. Vital signs and oxygenation results included HR of 88/min, BP of 112/78 mm Hg, SpO_2 of 96% on 35% F_IO_2 with satisfactory blood gases.

After satisfactory spontaneous parameters were performed (see above), she was extubated and placed on a nasal cannula at 3 L/min of O_2. She was weaned off oxygen and discharged from the hospital 6 days after admission.

Complications

The patient had no apparent pulmonary complications; however, there were minor neuromotor obstacles to overcome, including the loss of short-term memory and minimal motor sensory perception to the left. She will undergo physical and occupational rehabilitation after her release from the hospital.

Reference (hyperventilation to lower ICP):

Dumont, T. M. (2010). Inappropriate pre-hospital ventilation in severe traumatic brain injury increases in-hospital mortality. *Journal of Neurotrauma*, *27*(7), 1233–1241.

CASE 5: SMOKE INHALATION

INTRODUCTION

Patient history suggests potential complications of burn and smoke inhalation: infection, bronchospasm, carbon monoxide (CO), and cyanide poisoning.

F.H. was a 64-year-old, 76-Kg male admitted to the emergency department from his home where he was found in a smoke-filled room. He was unable to communicate with rescuers, appeared confused, and presented with first and second-degree burns over 50% of his upper torso. His facial features contained dark smoke and soot about his nose and mouth, a singed mustache, and he had a strong odor of alcohol on his breath.

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✓ Cyanide is one of the by-products of combustion.

The initial blood pressure was 139/100 mm Hg, temperature was 35.8°C with a pulse of 102/min, and respiratory frequency of 24/min while breathing on a non-rebreather mask at 15 L/min. He was immediately fluid-resuscitated with 1,000 mL of D_5W, and treated empirically for cyanide toxicity, which interferes with oxidative metabolism at the cellular level by impairing the utilization of oxygen in the tissues.

✓ With adequate spontaneous ventilation, the initial supportive measure for smoke inhalation is 100% O_2.

The clinical signs of cyanide poisoning at different toxic levels may include the following:

Blood cyanide concentration 0.2 to 0.3 mg/L	↑ HR, ↑ RR, dizziness
Blood cyanide concentration 0.3 to 1 mg/L	↑ Lethargy, arrhythmias, apnea
Blood cyanide concentration >1 mg/L	Death

✓ Pulse oximetry should not be used in smoke inhalation because it cannot distinguish carboxyhemoglobin from oxyhemoglobin and provides false high SpO_2 readings.

Upon admission into the hospital, the chest radiograph was normal (Figure 19-3). His breath sounds were mostly clear but dramatically changed following IV fluids to basilar crackles with expiratory wheezes throughout, and a prolonged expiratory phase. The patient maintained an SpO_2 of 99% and did not complain of dyspnea. A stat blood gas was ordered in the emergency room, followed immediately by a nebulizer treatment with 0.5 mL of 0.5% Proventil® in 2.5 mL of normal saline. The results of the blood gases were as follows:

✓ Blood gas samples must also be analyzed with carboxyhemoglobin (HbCO) in managing patients with smoke inhalation.

pH	7.30
$PaCO_2$	41 mm Hg
PaO_2	155 mm Hg
HCO_3^-	19.4 mEq/L
HbCO	21.2 g %
Hb	14.4 g %
CaO_2	12.7 vol %
SaO_2	92%
Mode	Non-rebreather mask
Flow	15 L/min

✓ In CO poisoning, the CaO_2 should be used to evaluate the patient's oxygenation status.

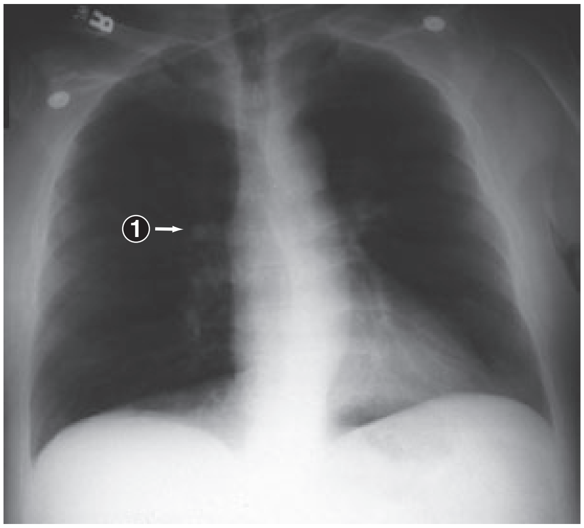

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FIGURE 19-3 Smoke inhalation. The chest radiograph is normal. The lungs typically are not affected unless there is edema formation secondary to smoke inhalation. The small round marking (1) represents a pulmonary blood vessel running parallel to the roentgen ray (X-ray).

✓ In spite of normal Hb and satisfactory SaO_2, the measured CaO_2 is low (12.7 vol %) because of the high HbCO level (21.2 g %).

Clinical symptoms generally arise with carboxyhemoglobin levels above 16 to 20%. These symptoms include headaches, dizziness, inability to concentrate, impaired judgment, lethargy, and confusion. Symptoms progressively increase between 20 and 60% HbCO, and exposures are fatal at 60 to 80% HbCO due to tissue asphyxiation. Lactic acidosis is a common sign that tissue oxygenation is impaired. Following a thorough assessment, the patient was then transported to the intensive care unit for further evaluation and treatment of his injuries.

Indications

✓ Elective intubation is done to maintain a patent airway in anticipation of airway edema and obstruction.

Physical examination revealed that the patient had received a moderate thermal burn to the airway as well as to his torso and extremities. He was electively intubated to maintain an adequate airway and, as expected, he became edematous to the point that his facial features were no longer recognizable.

Initial Settings

He was lightly sedated and placed on CPAP with pressure support of 15 cm H_2O, and the spontaneous frequency was 16/min. He maintained a spontaneous V_T of 550 mL (approx. 7 mL/Kg) at an F_IO_2 of 100% and PEEP of 6 cm H_2O. After 30 min, blood gases revealed:

✓ The PaO_2 (255 mm Hg) should not be used to guide oxygen therapy because dissolved oxygen contributes a very small fraction of arterial oxygen content.

✓ The measured CaO_2 showed improvement (from 12.7 to 14.7 vol %) after initiation of CPAP, PS, and 100% O_2.

pH	7.42
$PaCO_2$	36 mm Hg
PaO_2	255 mm Hg
HCO_3^-	22.8 mEq/L
HbCO	19.2 g %
Hb	14.4 g %
CaO_2	14.7 vol %
SaO_2	94%
Mode	CPAP
PEEP	6 cm H_2O
Pressure Support	15 cm H_2O
f (spontaneous)	16/min
V_T (spontaneous)	550 mL
F_IO_2	100%

The patient was maintained on his present settings because he had a normal acid-base balance and was breathing spontaneously. A high F_IO_2 was maintained to reduce the half-life of HbCO. The half-life of HbCO is as follows:

F_IO_2 100% at 3 atmospheric pressure	15 to 23 min
F_IO_2 of 100% and one atmospheric pressure	80 to 90 min
F_IO_2 of 21% (air) and one atmospheric pressure	280 to 320 min

Patient Monitoring

✓ HBO with 100% O_2 at 3 atm raises the dissolved oxygen to about 6 vol% - an amount that is sufficient to sustain cerebral metabolism.

Careful observation should be carried out to assess any evidence of pulmonary complications such as thermal injuries to the airway or pulmonary edema. These injuries may also increase the incidence and severity of ARDS as a complication to mechanical ventilation. Impaired gas exchange and/or hypoxia caused by increased levels of CO may be treated with PEEP or hyperbaric oxygen therapy (HBO) if CO poisoning is severe. The patient's mental functions should be monitored to prevent CO-induced anoxic brain syndrome.

Patient Management

The patient was closely monitored for 2 days while on mechanical ventilation for evidence of further injuries (i.e., decreasing lung compliance or evidence of ARDS, thermal airway injuries, and tissue hypoxia).

Key Medications

The patient was given Proventil® MDI treatments with 10 puffs inline through the ventilator circuit and continued with medication nebulizer treatments with Proventil® solution after extubation for wheezing.

Weaning

✓ Reduction of wheezing, improvement of breath sounds, and work of breathing are signs that the swelling to the airway has improved.

The swelling to the airway was significantly reduced by day 2 in the unit, and the patient was weaned and extubated after the return of adequate ventilation. His spontaneous frequency was 16/min, and he had adequate oxygenation and pulmonary mechanics. He was immediately given an aerosol treatment of 0.5 mL racemic epinephrine (Micronephrine®) in distilled water, to minimize stridor and reduce edema of the upper airway. Blood gases after extubation revealed the following:

pH	7.42
$PaCO_2$	38 mm Hg
PaO_2	104 mm Hg
HCO_3^-	23.6 mEq/L
HbCO	2.8 g %
Hb	14.1 g %
CaO_2	18.8 vol %
SaO_2	96%
Mode	Cool aerosol mask
f (spontaneous)	16/min
F_IO_2	28%

The patient remained in the hospital for three more days where he was weaned from the oxygen and required fewer medication nebulizer treatments to control his intermittent wheezing.

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Complications

The patient experienced no lasting complications from his injuries. He was weaned to room air and discharged from the hospital after day-6 postextubation.

CASE 6: DRUG OVERDOSE

INTRODUCTION

K.L. was a 78-year-old white male, 5 ft 8 in., medium build, who weighed about 73-Kg (160 lb). Upon admission to the 200-bed community hospital, his spontaneous frequency was 6/min and shallow. His skin color was pale but showed no cyanosis.

The paramedics stated that the patient was found by his neighbors in the bathroom lying on his back next to a pool of vomitus. The neighbors told the paramedics that the patient had a history of asthma and that he had been depressed since his girlfriend left him 2 weeks ago. An albuterol inhaler was found next to the patient and two empty bottles of tricyclic antidepressants were found on the kitchen table. Additional medical history revealed that the patient smoked about one pack of cigarettes each day.

The paramedics used a bag/mask resuscitator to ventilate the patient with O_2 at a frequency of about 20/min. They suctioned and removed large amounts of vomitus from his airway en route to the hospital.

Upon arrival at the emergency room, the respiratory therapist provided bag/mask ventilation. A nasogastric tube was inserted to prevent gastric distention and aspiration.

Cardiopulmonary assessments provided the following information: heart rate 45/min, weak pulses, systolic blood pressure 90 mm Hg, SpO_2 90%, spontaneous frequency 8/min, and shallow breath sounds with crackles and wheezes bilaterally.

Arterial blood gases on 100% O_2 revealed: pH 7.08, $PaCO_2$ 70 mm Hg, PaO_2 54 mm Hg, SaO_2 85%, and HCO_3^- 20 mEq/L.

✔ Blood gases show acute ventilatory failure with moderate hypoxemia.

Indications

The patient was intubated with a size 8.0 endotracheal (ET) tube. After checking for bilateral breath sounds, the ET tube was secured at the 24 cm mark at the lips. The end-tidal CO_2 reading was 20 mm Hg. During intubation, large amounts of watery brown secretions were suctioned from the airway and a sample was collected and sent to the laboratory for analysis.

✔ An end-tidal CO_2 reading of 20 mm Hg suggests correct placement of the ET tube in the trachea.

After the ET tube cuff was properly inflated, activated charcoal was put into the stomach via the nasogastric tube in order to absorb the remaining tricyclics.

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✓ Repeat suctioning is indicated because watery brown secretions (stomach content) have been suctioned from the ET tube.

While the activated charcoal was being administered, the airways and lungs were lavaged with normal saline and suctioned. This procedure was repeated until the return solution became clear.

A chest radiograph done later showed that the ET tube was 4 cm above the carina. Hazy infiltrates were noted in both lungs indicating that the patient had aspirated.

Another blood gas was drawn with the patient intubated and being bagged at a frequency of 20/min. The results were: pH 7.34, PCO_2 35 mm Hg, PO_2 350 mm Hg.

Initial Settings

✓ Since aspiration is likely to have occurred, PEEP of 5 cm H_2O is used to prevent atelectasis.

✓ Alarms should include low minute volume, low tidal volume, PEEP, high pressure, low pressure, high frequency, and apnea alarms.

The patient was transferred to the adult ICU and placed on volume-controlled ventilation. The initial settings were CMV mode, f 20/min, V_T 720 mL, F_IO_2 100%, PEEP 5 cm H_2O, peak flow 60 L/min, and constant flow pattern. With these settings, the peak inspiratory pressure was 25 cm H_2O, plateau pressure was 20 cm H_2O, and corrected V_T was 710 mL. All alarms were set appropriately. The ABG results on these settings were as follows:

pH	7.32
$PaCO_2$	32 mm Hg
PaO_2	264 mm Hg
HCO_3^-	16 mEq/L
SpO_2	98%
Mode	CMV
f	20/min
V_T	720 mL
F_IO_2	100%
PEEP	5 cm H_2O
PIP	25 cm H_2O
P_{PLAT}	20 cm H_2O

Patient Monitoring

A continuous SpO_2 monitor was placed on the patient with the low alarm set at 90%. A good waveform was noted, and the heart rate matched that on the cardiac monitor.

✓ Potential complications of aspiration may include aspiration pneumonia and ARDS.

✓ ARDS is usually preceded by increasing peak and plateau pressures (due to decreasing lung compliance), and increasing F_IO_2 requirement (due to intrapulmonary shunting).

Respiratory monitoring of the patient consisted of Q2° ventilator checks to include the peak inspiratory and plateau pressures, expired tidal volumes, minute volumes, F_IO_2, and compliance. Other clinical information and procedures included frequency, breath sounds, suctioning, and ET tube cuff pressure.

Due to the potential complications of aspiration, of particular importance was the monitoring of high peak and plateau pressure, F_IO_2, and PaO_2. A Swan-Ganz catheter and an arterial line were used to monitor the patient's hemodynamic status (i.e., arterial blood pressure, central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, cardiac output, and mixed venous saturation). Laboratory studies included serum electrolytes.

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Patient Management

Initially, the patient's respiratory status was stable but it began to deteriorate on the second day. The ventilator settings were changed to accommodate the patient's needs. The ventilator settings were: CMV mode, f 35/min, V_T 720 mL, F_IO_2 100%, 20 cm H_2O PEEP. The peak inspiratory pressure was 80 cm H_2O, and the plateau pressure was 70 cm H_2O. The blood gas results were:

pH	7.27
$PaCO_2$	43 mm Hg
PaO_2	72 mm Hg
HCO_3^-	19 mEq/L
SpO_2	92%
Mode	CMV
f	35/min
V_T	720 mL
F_IO_2	100%
PEEP	20 cm H_2O
PIP	80 cm H_2O
P_{PLAT}	70 cm H_2O

In PCV, the initial pressure may be set at 10 cm H_2O below the peak inspiratory pressure on CMV.

Because of the increasing pressure requirement, pressure-controlled ventilation (PCV) was started to minimize the occurrence of volume- or pressure-induced lung injuries. The pressure on PCV was initially set at 70 cm H_2O (10 cm H_2O below the peak inspiratory pressure on CMV). Other ventilator parameters for PCV were: inspiratory time 1 sec, f 35/min, F_IO_2 100%, 20 cm H_2O PEEP. The blood gases on these settings were as follows:

pH	7.31
$PaCO_2$	41 mm Hg
PaO_2	56 mm Hg
HCO_3^-	20 mEq/L
SpO_2	88%
Mode	PCV
PIP	70 cm H_2O
f	35/min
F_IO_2	100%
PEEP	20 cm H_2O

In PCV, the minute volume is primarily determined by the pressure, inspiratory time, and lung compliance.

In ARDS, a lung protection strategy includes use of low pressure and volume.

The inspiratory time was then increased to 2 sec to keep the PaO_2 in the 70s. The I:E ratio was changed to 4:1 to provide longer inspiratory time. Subsequently, the minute volume was increased with these settings and the pressure was lowered to 50 cm H_2O. Blood gases on these settings were satisfactory. The minute volume of the patient was trended carefully during the use of PCV.

The patient was placed on a Roto-bed and was administered neuromuscular and sedative agents to minimize agitation and oxygen consumption. For proper airway management, a tracheostomy was performed because extended mechanical ventilation was anticipated.

Permissive hypercapnia is used to reduce the pressure and volume requirement for ventilation.

To further minimize pressure-induced lung injuries, the pressure on PCV was further decreased until the tidal volume was about 450 mL. The $PaCO_2$ was allowed to reach 80 mm Hg (permissive hypercapnia) while the pH was maintained near 7.35 by bicarbonate infusion and kidney compensation.

Key Medications

No medication was needed for intubation because the patient was unconscious at the time of intubation. Additional medications were withheld because of the uncertain drug interaction with other unknown drugs that the patient might have taken.

The patient was given neuromuscular blocking agents and sedatives while on mechanical ventilation. As the respiratory status improved, the patient was weaned from these drugs. Albuterol was given prn for wheezing.

Weaning

Over the next 2 weeks, the patient continued to improve. Weaning was done by alternating CPAP with pressure support (during day) and SIMV (during night). Both modes of ventilation were supported with flow triggering. The SIMV mode allowed the patient to rest. He was also ambulated daily to strengthen his respiratory muscles and exercise endurance.

MIP > -30 cm H_2O and f/V_T ratio of less than 100/min/L correlate with weaning success.

As he regained strength, the following spontaneous respiratory parameters were obtained: f <35/min, minute volume around 9 L, vital capacity >1 L, MIP >-30 cm H_2O, and $f/V_T < 100$/min/L.

Eventually, the patient was weaned to CPAP of 5 cm H_2O, F_IO_2 35%, and pressure support of 5 cm H_2O. A spontaneous frequency of 16 to 20/min yielded these ABG results:

pH	7.37
$PaCO_2$	43 mm Hg
PaO_2	80 mm Hg
HCO_3^-	24 mEq/L
SpO_2	95%
Mode	CPAP
PEEP	5 cm H_2O
F_IO_2	35%
PS	5 cm H_2O
Spont f	16 to 20/min

After 2 days of CPAP, the patient continued to improve, he was placed on a nasal cannula at 4 L/min and his fenestrated tracheostomy tube was buttoned, allowing the patient to breathe through his upper airway. He tolerated the closure of his tracheostomy very well, and it was left this way for 2 days. Two days later, he was transferred to the rehabilitation floor.

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✓ Tension pneumothorax was probably caused by excessive airway and pulmonary pressures.

✓ To prevent self-extubation, neuromuscular blocking agents and sedatives may be used to provide comfort. An alternative is to use active restraints with frequent assessment for continuing use.

Complications

During the course of mechanical ventilation, a left-sided tension pneumothorax occurred and this led to decreased lung compliance, decreased O_2 saturation, and decreased breath sounds on the left side. Chest radiography showed a shift of the mediatinum to the right side. The tension pneumothorax gradually resolved with the placement of a chest tube.

At one point during weaning the patient became very combative and he self-extubated. He was reintubated without delay. Cloth restraints were then used to secure his extremities.

CASE 7: TENSION HEMOPNEUMOTHORAX

INTRODUCTION

✓ A chest tube is used to remove air, blood, or fluid accumulated in the pleural space.

✓ Hypotension and tachycardia are two common signs of inadequate blood volume.

✓ Tension pneumothorax or hemopneumothorax shifts the mediastinum to the unaffected (opposite) side.

✓ Colloids are used to enhance fluid balance in the early phase of volume replacement.

P.S. was a 50-year-old, 96-Kg male transferred from another hospital with a complicated, right lower lobe pneumonia and hemothorax. At the onset, a small right pleural effusion was found that required drainage of 600 mL exudates material via a needle thoracentesis. This effusion again re-accumulated and a second thoracentesis was performed that was productive of over 400 mL of thin, cloudy fluid. A chest tube was placed following the procedure, which continued to drain thin, cloudy fluid followed by frank blood. This output continued throughout the day, requiring massive transfusions to maintain his blood pressure; subsequently, he was transferred to a tertiary care facility for definitive therapy. Prior to air transport, the patient was volume-resuscitated with both blood and fluid, nasally intubated, and noted to have inadequate drainage of the right hemothorax.

Upon arrival at the ICU, the patient developed further hemodynamic instability with a systolic pressure of 70 mm Hg (*normal 120 mm Hg*) and a heart rate of 120/min (*normal 60 to 100/min*). He had weak peripheral pulses, dullness to percussion of the right thorax, expiratory wheezes, and coarse rhonchi, with the left being greater than the right. The chest tube drained about 950 mL of bloody fluid. The patient was resuscitated with colloids followed by more blood transfusions. A chest radiograph revealed a complete opacification on the right with concurrent mediastinal shift to the left side (Figure 19-4).

Indications

The patient was electively intubated prior to transport, to establish and maintain an adequate airway. Mechanical ventilation was established to secure an airway prior

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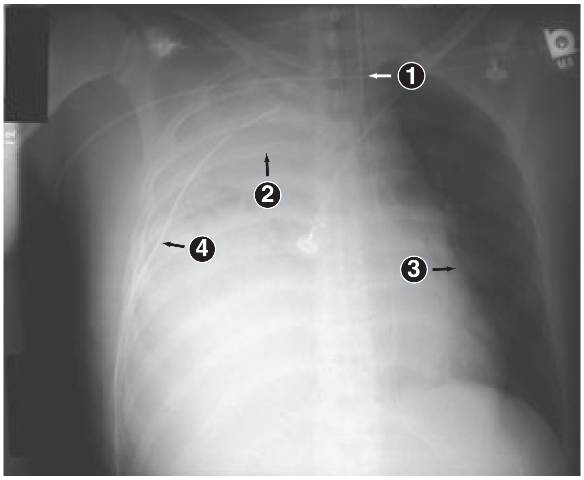

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FIGURE 19-4 Tension hemopneumothorax. Right-sided hemopneumothorax shifts the mediastinum and trachea (1) to the left. The shift is not significant, possibly due to patient rotation when the radiograph was taken. The white area (2) on the radiograph is caused by the blood in pleural space. (*Note: air in pleural space would appear dark.*) Compression of the left lung is noted (3). Chest tube (4) can be seen on the right side.

to flight because of hemodynamic instability, reduced lung volumes, and an apparent increase in the work of breathing.

Hemodynamic instability is mainly due to blood loss.

Initial Settings

The patient was lightly sedated and placed on volume-controlled ventilation in the assist/control mode at 16/min with V_T of 800 mL (approx. 8 mL/Kg), F_IO_2 of 100%, and PEEP of 5 cm H_2O. There was no spontaneous respiratory effort.

Reduction of lung volumes is caused by compression of the lungs by blood and air in the pleural space.

The peak inspiratory pressures exceeded 70 cm H_2O with each breath, and the tidal volume delivered was about 8 mL/kg of body weight. This relatively low volume maintained adequate ventilation without excessive cardiovascular compromise induced by positive pressure ventilation.

The patient was hemodynamically stabilized using stored blood and clinically evaluated with a chest radiograph and arterial blood gas analysis. The initial blood gas results were as follows:

Increase in work of breathing is caused by reduction of lung compliance and/or increase of airway resistance.

pH	7.30
$PaCO_2$	40 mm Hg
PaO_2	83 mm Hg
HCO_3^-	18.9 mEq/L
B.E.	−6.8 mEq/L
Hb	15.8 g %
CaO_2	20.9 vol %
SaO_2	94%
SpO_2	89%
Mode	A/C
f	16/min

The peak inspiratory pressure was high (>70 cm H_2O) because of low lung compliance (due to compression of lung parenchyma by blood and air in the pleural space).

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V_T	800 mL
F_IO_2	100%
PEEP	5 cm H_2O

Twenty-five minutes later, the patient remained in critical but stable condition while continuing to bleed into the chest tube. The endotracheal tube position was noted to be at the 24 cm mark at the lip and secured in the airway, awaiting confirmation of tube position on the chest radiograph. Blood gases were drawn to evaluate his acid-base status and to quantify the degree of blood loss from the tension hemothorax. These were outlined as follows:

Change in the hemoglobin level (from 15.8 to 10.7 g %) greatly diminishes the patient's arterial oxygen content (from 20.9 to 15 vol %), confirming the presence of tissue hypoxia.

pH	7.40
$PaCO_2$	33 mm Hg
PaO_2	196 mm Hg
HCO_3^-	19.6 mEq/L
B.E.	−4.2 mEq/L
Hb	10.7 g%
CaO_2	15 vol%
SaO_2	96%
SpO_2	95%
Mode	A/C
f	16/min
V_T	800 mL
F_IO_2	100%
PEEP	5 cm H_2O

The F_IO_2 was reduced to 60% to lower the PaO_2 while being mindful of the low oxygen content. A chest radiograph confirmed proper placement of the endotracheal tube and noted a tension hemothorax on the right side. He was taken to the operating room for an emergency thoracotomy and relief of the tension hemothorax resulting from a lacerated intercostal vessel.

The F_IO_2 was reduced to 60% because the PaO_2 (196 mm Hg) was excessive.

By the end of the thoracotomy and repair of the bleed, he received 11 units of blood prior to returning to the ICU. Later, he received 9 more units of blood in the ICU.

Patient Monitoring

Upon return from the operating room (OR), the patient was placed back on mechanical ventilation at 1,000 mL tidal volume (approx. 11 mL/Kg) on assist/control (A/C) with a frequency of 12/min, F_IO_2 of 60%, and 5 cm H_2O of PEEP. Peak inspiratory pressures returned to 36 cm H_2O. The patient's hemodynamic status was stable and he was allowed to awaken from the anesthesia. Blood gases 10 min after returning from surgery revealed the following:

pH	7.41
$PaCO_2$	39 mm Hg
PaO_2	55 mm Hg
HCO_3^-	24.1 mEq/L

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B.E.	−2.7 mEq/L
Hb	11.4 g%
CaO_2	17.7 vol%
SaO_2	90.5%
SpO_2	87%
Mode	A/C
f	12/min
V_T	1,000 mL
F_IO_2	60%
PEEP	5 cm H_2O

In an effort to minimize the effects of oxygen toxicity, the ventilator was adjusted by increasing the PEEP to 8 cm H_2O to increase the PaO_2 without raising the F_IO_2 over 60%.

Patient Management

✓ Cardiac output and pulmonary capillary wedge pressures are used to monitor the adequacy of circulating blood volume.

This patient was recovered from surgery in the ICU on mechanical ventilation, in an effort to minimize any postsurgical complications associated with continued blood loss, and to guard against the development of respiratory distress.

Following the rapid infusion of over 20 units of blood to cover his losses, assessments with hemodynamic measurements including cardiac output and pulmonary capillary wedge pressures (PCWP) helped to assure adequate perfusion to the tissues. Oxygenation, under the conditions presented, may be complicated by the use of stored blood used for volume replacement. Of particular importance is the fact that stored blood contains limited amounts of 2,3 diphosphoglycerate (DPG), which effectively shifts the oxyhemoglobin dissociation curve to the left. Thus, stored blood reduces the unloading of oxygen at the tissues, causing hypoxia, necrosis, and possibly sepsis if not managed properly. As a result, the patient's PaO_2 was closely monitored to maintain adequate oxygenation and saturation. He was also monitored for clinical signs of tissue hypoxia with arterial-venous oxygen content difference [$C_{(a\text{-}v)}O_2$]. At that time, follow-up blood gases revealed these results:

pH	7.47
$PaCO_2$	34 mm Hg
PaO_2	124 mm Hg
HCO_3^-	24.1 mEq/L
B.E.	−1.7 mEq/L
Hb	11.4 g%
CaO_2	15.5 vol%
SaO_2	95.7%
SpO_2	94%
Mode	A/C
f	12/min
V_T	1,000 mL
F_IO_2	60%
PEEP	8 cm H_2O

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✓ Reduce F_IO_2 when PaO_2 is too high.

The F_IO_2 was reduced to 50% and the patient was continuously monitored by the SpO_2 and SvO_2 measurements.

Respiratory Care Procedures

Based on the preadmission diagnosis of complicating pneumonia, the patient was aggressively treated with frequent suctioning, with moderate amounts of cloudy secretions removed.

Bronchopulmonary toilet was initiated, and the patient was immediately started on bronchodilator therapy for wheezing, with 20 puffs of Proventil® via a metered-dose inhaler (MDI) given inline through the ventilator circuit via Aerochamber®, and he was frequently turned from side to side to help prevent atelectasis.

NOTE: Clinical use and evaluation of lavage must be carefully considered; i.e., see Pedersen, C. R. (2009). Endotracheal suctioning of the adult intubated patient--what is the evidence? *Intensive and Critical Care Nursing*, *25*(1), 21–30. Ackerman, M. H. (1993). The effect of saline lavage prior to suctioning. *Journal of Critical Care, 2*(4), 326–330.

Weaning

Two days following surgery, the patient began to breathe spontaneously and he was changed to SIMV in an attempt to wean him from the ventilator. Initially, his frequency was decreased to 8/min, and all spontaneous breaths were augmented by 10 cm H_2O of pressure support. He was able to initiate 20 breaths above the set frequency and maintained a tidal volume of 400 to 600 mL at this level of support. His ventilator tidal volume was set at 1,000 mL, and over that time his PEEP was reduced to 5 cm H_2O and his F_IO_2 was reduced to 40%. As his muscular effort improved (as evidence revealed that he was able to maintain tidal volume at progressively lower pressure support settings), the tidal volume increased for spontaneous breaths, and his pressure support was quickly weaned to 6 cm H_2O. He remained at that level throughout the day without signs of fatigue, hypoxemia, tachypnea, hypertension, desaturation, or evidence of tachycardia.

That night he was placed on assist/control in an effort to rest the muscles of inspiration. Weaning began at six o'clock the next morning on CPAP with pressure support of 12 cm H_2O, PEEP of 5 cm H_2O, and F_IO_2 of 40%. Spontaneous parameters obtained 2 hours later revealed:

✓ MIP of -52 cm H_2O and f/VT of <100/min/L correlate with weaning success.

$f = 26 \quad V_E = 12.4\text{ L} \quad V_T = 0.47\text{ L} \quad VC = 1.09\text{ L}$
$MIP = -52\text{ cm }H_2O \quad f/V_T = 55\text{/min/L}$

Based on stable spontaneous parameters and improving clinical condition, he was removed from the ventilator and allowed to breathe on a "T-piece" at 40% F_IO_2 for 4 hours. Subsequent evaluations led to successful weaning and eventual extubation. He was placed on a nasal cannula at 6 L/min where he continued to improve until discharge.

Complications

Besides the tension hemopneumothorax, there were no apparent complications from either the right lower lobe pneumonia or throughout the postoperative period resulting from the mechanical ventilation. He was followed throughout his hospital stay with respiratory therapy and continued treatments with deep breathing (IS) regimens and with bronchodilator therapy for wheezing. His oxygen demands were monitored with daily pulse oximetry and the oxygen flow was titrated to keep the SpO_2 above 90%. He was discharged on the eighth postsurgical day from the tertiary care facility on room air and without further complications.

CASE 8: CHEST TRAUMA

INTRODUCTION

A.P. was a 24-year-old, 56-Kg female involved in a moving vehicle accident. The patient was unrestrained and was struck on her side of the vehicle. She was thrown from the vehicle, and her car was found to contain multiple prescription pain medication bottles. She was apparently comatose at the scene with no spontaneous breathing or pulse. However, her vital signs returned to normal en route to the hospital.

Upon arrival at the emergency department, her vital signs revealed a blood pressure (BP) of 122/80 mm Hg, pulse of 80/min, normal sinus rhythm, and temperature of 35.8°C. Her breathing was assisted by the respiratory care practitioner (RCP) with a manual resuscitator bag. Breath sounds revealed diffuse coarse rhonchi bilaterally without wheezing.

✓ The white blood count is significantly elevated.

Laboratory and radiology results revealed a white blood cell (WBC) count of 17.5×10^3 (*normal* 3.2 *to* 9.8×10^3), hemoglobin (Hb) of 12.7 g% (*normal 12 to 15 g %*), platelets of 195×10^3 (*normal* 130 *to* 400×10^3), prothrombin time (PT) of 13 sec (*normal 9 to 12 sec*), and a partial thromboplastin time (PTT) of 30 sec (*normal 22 to 37 sec*). A portable chest radiograph (Figure 19-5) revealed rib fracture on the right and bilateral infiltrates in which the left side was greater than the right. The left hemidiaphragm, heart border, and parenchymal changes were consistent with pulmonary contusion and/or possible aspiration pneumonitis.

✓ Pulmonary contusion is an internal injury of the lung parenchyma in which the skin is not broken.

✓ Bilateral rib fractures and pneumothoraces with blood accumulation limit chest expansion and hinder ventilation and oxygenation.

The patient underwent an extensive evaluation of her injuries, including a computerized tomography (CT) of her head that was essentially normal. Scans of her chest and abdomen revealed bilateral rib fractures, pneumothoraces with blood accumulation, and rupture of her spleen with evidence of free peritoneal fluid. Also noted was an apparent transverse process fracture of the lumbar spine as well as probable posterior element fractures of the T1 vertebrae. She also had multiple contusions and lacerations, including a large laceration to the right axilla and in the right wrist region.

Her previous history included smoking one pack of cigarettes per day and chronic bronchitis.

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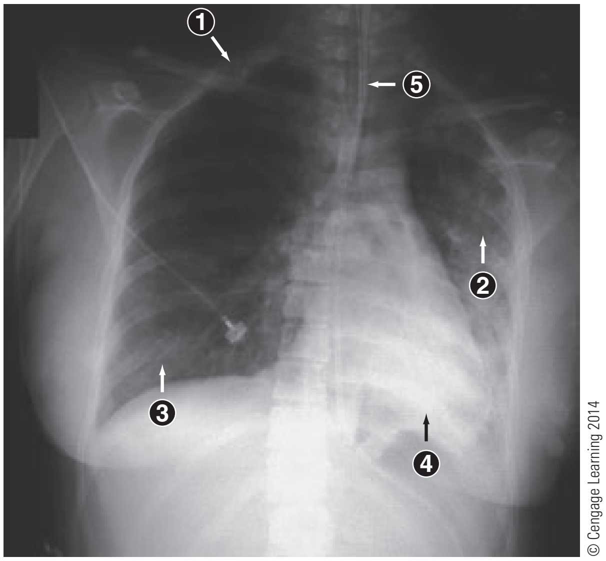

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FIGURE 19-5 Chest trauma. Rib fracture is noted on the right side (1) of this chest radiograph. Infiltrates of the left side (2) cause a greater density than the right side (3). The diaphragm shadow on the left is lost (4) likely due to pulmonary contusion or possible aspiration pneumonitis. Left-sided atelectasis pulls the mediastinum and trachea (5) to the left. The shift does not appear to be significant, possibly due to patient rotation when radiograph was taken.

Indications

✔ Chest trauma, and unstable and worsening cardiopulmonary and hemodynamic status are the primary indications for mechanical ventilation.

Due to the extent of her injuries, she was hemodynamically stabilized with a transfusion of four units of blood. A short-acting neuromuscular blocker (Succinylcholine) was used to facilitate intubation with a size 8.0 endotracheal tube. She was subsequently transferred to the intensive care unit (ICU) in critical but stable condition requiring mechanical ventilation.

Initial Settings

✔ A $PaCO_2$ of 25 mm Hg shows alveolar hyperventilation, suggesting ventilatory insufficiency (i.e., hyperventilation to maintain a PaO_2 of 83 mm Hg) on 60% F_IO_2.

She was immediately placed on volume-controlled ventilation on assist/control at 15/min V_T 600 mL (approx. 10 mL/Kg), F_IO_2 60%, and PEEP 5 cm H_2O. Chest tubes were placed to evacuate the pleural space of blood and air. Her initial blood gases after 10 minutes on the ventilator were:

pH	7.46
$PaCO_2$	25 mm Hg
PaO_2	83 mm Hg
HCO_3^-	17.3 mEq/L
Hb	13.7 g %
Mode	A/C
f	15/min
V_T	600 mL
F_IO_2	60%
PEEP	5 cm H_2O

✔ Prolonged hyperventilation leads to patient fatigue and deterioration of ventilation and oxygenation status.

Over the next 24 hours, her condition quickly deteriorated, requiring 100% F_IO_2 and increasing levels of PEEP to maintain adequate oxygenation.

Patient Monitoring

✓ Pressure Control-Inverse Ratio Ventilation (PC-IRV) uses a predetermined inspiratory pressure to minimize the occurrence of pressure-induced lung injury. Use of inverse I:E ratio is intended to increase inspiratory time and lung expansion, and to improve oxygenation.

✓ PC-IRV increases the mean airway pressure, central venous pressure, and pulmonary artery pressure.

The increasing pressure required to ventilate her lungs began to compromise her cardiac output and, as such, limit perfusion to her tissues. As a result, she was deeply sedated and changed to *pressure-controlled inverse ratio ventilation* (PC-IRV) *mode* at a frequency of 12/min and an inspiratory pressure of 30 cm H_2O. Her inspiratory time (T_I) was adjusted accordingly to obtain an I:E ratio of 3:1. Her PEEP and F_IO_2 requirements were 8 cm H_2O and 100%, respectively. These settings provided hyperexpansion of the chest in order to stabilize the thorax and limit movement of the rib fractures. She was monitored by an end-tidal CO_2 monitor. Frequent blood gases were done to evaluate her ventilatory and oxygenation status. Blood gases drawn at this time revealed the following:

pH	7.36
$PaCO_2$	42 mm Hg
PaO_2	62 mm Hg
SaO_2	90%
HCO_3^-	22.9 mEq/L
Hb	11.2 g %
Mode	PC-IRV
T_I	1.0 sec
I:E ratio	3:1
f	12/min
P_{INSP}	30 cm H_2O
F_IO_2	100%
PEEP	8 cm H_2O

✓ PC-IRV can cause hemodynamic instability.

The patient appeared to tolerate these changes well. However, within 1 hour her hemodynamic values (including cardiac output) were reduced as a result of pressure ventilation. The T_I was decreased to 0.82 sec (2:1 ratio) and frequency was increased to 24/min. The PEEP was increased to 15 cm H_2O. Arterial and mixed venous blood gases revealed the following:

Arterial

pH	7.38
$PaCO_2$	38 mm Hg
PaO_2	90 mm Hg
SaO_2	95%
HCO_3^-	21.8 mEq/L
B.E.	−2.4 mEq/L
Hb	11.1 g%
CaO_2	14.9 vol%

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Mixed Venous

pH	7.36
PvO_2	47 mm Hg
SvO_2	78%
$A\text{-}aDO_2$	462 mm Hg
Qs/QT	42%

Ventilator Settings

Mode	PC-IRV
T_I	0.82 sec
I:E ratio	2:1
f	24/min
PIP	30 cm H_2O
F_IO_2	100%
PEEP	15 cm H_2O

✔ Ventilator settings should be adjusted based on the patient's ventilation and oxygenation requirement, as well as on the patient's hemodynamic status.

The patient remained on a 2:1 I:E ratio, and other ventilator settings were adjusted to normalize ventilation and oxygenation. To maintain a PaO_2 greater than 65 mm Hg ($SpO_2 > 90\%$), the required settings were: peak inspiratory pressure 30 cm H_2O, PEEP 18 cm H_2O, and F_IO_2 70%.

Patient Management

✔ Aggressive pulmonary hygiene is important in managing patients who are intubated and receiving prolonged mechanical ventilation.

For the next 14 days, the patient was monitored closely for adverse signs while in a drug-induced coma. She was placed on a proprofol (Diprivan®) drip to manage her pain and was given narcotic medications to limit her movements while on pressure-controlled ventilation. Vigorous pulmonary toilet was begun and she was suctioned with a closed directional-tip catheter to the main-stem bronchi. This produced a large amount of thick, yellow secretions. She also received frequent acetylcystine (Mucomyst) lavage every 4 hours and was confined to a Rotorest® bed to help prevent pulmonary complications associated with atelectasis.

NOTE: Clinical use and evaluation of lavage must be carefully considered i.e., see Pedersen, C. R. (2009). Endotracheal suctioning of the adult intubated patient—what is the evidence? *Intensive and Critical Care Nursing, 25*(1), 21–30. Ackerman, M. H. (1993). The effect of saline lavage prior to suctioning. *Journal of Critical Care, 2*(4), 326–330.

Key Medications

She was given nebulizer treatments with 0.5 mL of 0.5% albuterol sulfate (Proventil®) and 0.5 mg ipratropium bromide (Atrovent®) every 4 hours for wheezing. Broad-spectrum antibiotics including a fourth-generation cephalosporin were used to treat ventilator-associated pneumonia (VAP) from long-term ventilation.

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Weaning

✓ For ventilator patients requiring high F_IO_2 and PEEP, wean F_IO_2 to about 40% before weaning PEEP.

Several days later, the patient's oxygen was weaned to 45% and frequency of 22/min. Her condition continued to improve over time and the RCPs were also able to wean the PEEP to 15 cm H_2O. Blood gases on these settings revealed:

pH	7.58
$PaCO_2$	26 mm Hg
PaO_2	87 mm Hg
SaO_2	96%
HCO_3^-	23.5 mEq/L
B.E.	3.3 mEq/L
Hb	11.7 g %
CaO_2	15.8 vol %
Mode	IR-PCV
f	22/min
PIP	30 cm H_2O
F_IO_2	45%
PEEP	15 cm H_2O

At this point, she was removed from pressure-controlled ventilation and allowed to awaken. As her spontaneous breathing resumed, she was quickly weaned to continuous positive airway pressure (CPAP) with 5 cm H_2O of PEEP and pressure support of 20 cm H_2O without difficulty. She maintained a frequency of less than 24/min with tidal volumes of about 300 mL. As her muscular effort improved, tidal volume increased and her pressure support was titrated to maintain the same tidal volume and assure adequate alveolar ventilation and lung expansion.

✓ During a weaning attempt, the pressure support level should be titrated to obtain a desired spontaneous V_T or f.

At night, she was rested on assist/control at a V_T of 600 mL, backup frequency of 12/min. She maintained a spontaneous frequency of 14/min. This regimen continued for the next 3 days where she was placed on CPAP with pressure support titrated to assist her spontaneous tidal volumes during the day, and rested during the night. Spontaneous parameters were obtained each morning to determine suitability to wean and monitor her effort to breathe on her own. Eventually, rather than returning to assist/control, she was able to tolerate synchronized intermittent mandatory ventilation (SIMV) mode with pressure support of 15 cm H_2O during the night. The following day, she remained on CPAP with 20 cm H_2O of pressure support throughout the next 36 hours, without signs of respiratory insufficiency or distress.

She was evaluated for extubation, and her spontaneous respiratory parameters over the last 5 days were:

Day 1: $f = 50$ $V_E = 14.8$ L $V_T = 0.3$ L VC = 0.80 L
MIP = −46 $f/V_T = 167$

Day 2: $f = 40$ $V_E = 10.6$ L $V_T = 0.27$ L VC = 0.85 L
MIP = −48 $f/V_T = 148$

Day 3: $f = 37$ $V_E = 9.5$ L $V_T = 0.26$ L VC = 0.87 L
MIP = −56 $f/V_T = 142$

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Day 4: f = 33 V_E = 8.2 L V_T = 0.3 L VC = 1.02 L
MIP = −57 f/V_T = 110

Day 5: f = 32 V_E = 10.3 L V_T = 0.32 L VC = 1.14 L
MIP = −42 f/V_T = 100

✓ f/V_T of 100/min/L or *lower* suggests readiness for weaning attempt. Rapid shallow breathing usually results in a *high* f/V_T.

✓ An f/V_T ratio of less than 100 is predictive of weaning success.

Extubation decisions are frequently made in conjunction with spontaneous breathing parameters as well as other clinical indicators that may predict success. Ideally, every member of the patient care team may give input concerning this important decision. The frequency (f) should be below 30/min. The minute volume (V_E) should be around 10 L with a tidal volume of >5 to 8 mL/kg and a vital capacity >10 to 15 mL/kg. The maximum inspiratory pressure (MIP) should be greater than −20 to −30 cm H_2O where muscle strength is directly proportional to the volume generated for inspiration. The frequency to tidal volume ratio (f/V_T), sometimes called rapid shallow breathing index (RSBI), measures the patient's volume as compared to the frequency. Patients breathing rapidly with shallow volumes may only ventilate the anatomic deadspace (i.e., conducting zones within the lungs without effective alveolar ventilation). This ratio, then, should be below 100/min/L before effective weaning may take place.

Realize, however, that these indicators are only idealistic values and should not be taken individually as predictors of success. Other clinical signs such as level of consciousness, sensorium, ability to follow commands, ability to lift head off pillow, character and volume of secretions, and hemodynamic status may also help in predicting a successful postextubation course.

Complications

The patient followed a gradual resolution of her multiorgan dysfunction and severe refractory pulmonary difficulties after an 18-day ventilator course. She was recovered and discharged to a home health agency where she was advised to abstain from prescription narcotics, continued on Gentamicin 360 mg IV once per day, and Ciprofloxacin 750 mg twice daily for 10 days. Her SpO_2 at discharge was 90% on room air while walking.

CASE 9: ACUTE RESPIRATORY DISTRESS SYNDROME

INTRODUCTION

M.I. was a 22-year-old, 54-Kg female with a history of systemic lupus erythematosus, congenital mitral regurgitation, and syncope. For the past several months, she complained of a mild productive cough while lying down. One week prior to her admission she developed a severe cough, progressive dyspnea without the use of accessory muscles, and associated pain throughout her chest wall and into her upper abdomen.

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An iron supplement is used to increase the oxygen-carrying capacity of hemoglobin.

Hemoglobin, WBC, HCT, and blood pressure are all outside normal limits.

Her medical history also included chronic anemia (hemoglobin 8.7 g %, *normal 12 to 15 g %*) and was on an iron supplement at the time. Her white blood cell (WBC) count was 21 × 10^3 (*normal 3.2 to 9.8 × 10^3*), hematocrit (HCT) 27% (*female average 42%*) without evidence of blood loss. Blood pressure was 93/59 mm Hg (*normal 120/80 mm Hg*), HR 114/min, and spontaneous respirations were shallow and guarded at 20/min. Tenderness was noted across the upper abdomen and the lower ribs without organomegaly or other masses present.

She was 4 months postpartum with gradual onset abdominal pain, progressive dyspnea with a nonproductive cough, and was admitted to the hospital for further evaluations.

Persistent hyperventilation ($PaCO_2$ 31 mm Hg) with moderate hypoxemia (PaO_2 67 mm Hg) could lead to fatigue of respiratory muscles if causes of hypoxemia are not identified and treated promptly.

The patient's nonspecific symptoms led to a wide range of diagnostic tests for her condition, which included lupus, mitral regurgitation, and pulmonary insufficiency. Initial blood gases on room air were obtained in the emergency room, the patient was admitted to the medical floor, and a sputum culture was ordered to evaluate pathology concerning the respiratory tract.

The initial blood gases were as follows:

pH	7.49
$PaCO_2$	31 mm Hg
PaO_2	67 mm Hg
HCO_3^-	22.7 mEq/L
Hb	8.4 g %
Mode	Spontaneous
F_IO_2	21%

Her breath sounds were unremarkable but she was moderately fatigued and continued to be short of breath. She was started on a nasal cannula at 2 L/min of oxygen and was encouraged to deep-breathe and cough. At this point, the patient was still able to adequately ventilate as evidenced by the $PaCO_2$ of 31 mm Hg. However, her condition deteriorated during the course of her evaluation and workup. Shortly thereafter, she developed intermittent wheezing for which albuterol nebulizer treatments were administered.

Indications

Impending ventilatory failure typically shows increasing $PaCO_2$ and decreasing pH and PaO_2.

Her blood gases continued to deteriorate and on day 6, she required a non-rebreather mask at high oxygen flow to maintain her SpO_2 above 90%. She showed signs of impending ventilatory failure evidenced by the rising $PaCO_2$ and acidotic pH on subsequent blood gases. A chest radiograph showed areas of bibasilar atelectasis and haziness with bilateral infiltrates. No pulmonary consolidation was noted. Postural drainage and chest physiotherapy were started to help facilitate removal of secretions, but she was too weak to generate a productive cough and unable to contribute to her pulmonary care.

Postural drainage and chest physiotherapy were done to facilitate loosening and removal of secretions.

Family members refused permission to obtain a diagnostic bronchoscopy or any attempts at percutaneous biopsies to evaluate lung pathology. Three days later, her oxygen requirements had increased to the point where she required a heated nebulizer analyzed at an F_IO_2 of 95% bled into her non-rebreather mask at 15 L/min,

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and a nasal cannula at 6 L/min simply to maintain oxygen saturation of 86% to 92% without activity. Every effort was made to forestall clinical deterioration within the treatment parameters that family members permitted. Practitioners were able to convince the family to utilize free-standing CPAP at 12 cm H_2O and F_IO_2 of 50% to improve oxygenation, but the patient became fatigued to the extent that she was only able to tolerate the procedure 20 minutes every 2–3 hours, if at all. At that time she had a vital capacity of 0.9 L and MIP of −75 cm H_2O. This treatment continued for 2 days as she became refractory to oxygen therapy and was finally unable to maintain adequate oxygen saturation. Her chest radiograph revealed patterns of homogenous densities (i.e. uniform reticulogranular patterns or ground glass appearance) in both lung fields. This sign is indicative of the development of acute respiratory distress syndrome (ARDS).

✓ CPAP requires adequate spontaneous breathing. Development of respiratory muscle fatigue is the primary concern for prolonged CPAP usage in critically ill patients.

Initial Settings

Every attempt was made to avoid ventilatory failure and prevent intubation, but the patient's condition continued to deteriorate, and she became progressively fatigued. Her age and previous health were positive factors in postponing ventilatory failure, but she eventually became overwhelmed by fatigue. She was moderately sedated, intubated with a size 7.5 endotracheal tube, and placed on volume-controlled ventilation on assist/control at a frequency of 25/min, V_T of 600 mL (approx. 11 mL/Kg), F_IO_2 of 100%, and PEEP of 8 cm H_2O. Arterial and mixed venous blood gases on these settings revealed the following:

Arterial

pH	7.57
$PaCO_2$	33 mm Hg
PaO_2	58 mm Hg
SaO_2	88%
HCO_3^-	29.5 mEq/L
Hb	10.1 g %

Mixed Venous

pH	7.38
PvO_2	33 mm Hg
SvO_2	58%
$C(a\text{-}v)O_2$	4.6 vol %
Qs/QT	37%

Ventilator Settings

Mode	A/C
f	25/min
V_T	600 mL
F_IO_2	100%
PEEP	8 cm H_2O

✓ The ventilator frequency was decreased because of the low $PaCO_2$ (33 mm Hg).

✓ The PEEP was increased because of the low PaO_2 (58 mm Hg) in spite of 100% F_IO_2.

Her ventilator frequency was reduced to 14/min, and her PEEP was increased to 10 cm H_2O.

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Patient Monitoring

✓ With cardiogenic pulmonary edema, the PCWP is usually elevated (>18 mm Hg).

✓ Pulmonary edema with normal PCWP is likely caused by lung parenchymal changes (e.g., ARDS).

In addition to blood gases and vital signs, other cardiopulmonary monitoring included chest radiography and hemodynamic measurements. Her chest radiograph at that time revealed initial findings consistent with ARDS including a uniform reticulogranular pattern or ground glass appearance throughout both lung fields. Arterial and pulmonary artery catheters were used to continuously monitor blood pressure, cardiac output, and hemodynamic status. Cardiac output was stable at 4.2 L. The pulmonary capillary wedge pressure (PCWP) was around 11 mm Hg and thus ruled out cardiogenic pulmonary edema. The development of refractory hypoxemia in ARDS may be summarized as follows:

> Hypoxic vasoconstriction in ARDS increases the vascular resistance in the lungs and can diminish cardiac output. The lack of oxygen in the pulmonary circulation also causes necrosis of the tissue lining of the alveolar-capillary membrane which, in turn, induces pulmonary capillary leak into the interstitium (third spacing) and hence, impairs gaseous exchange with the blood. This eventually causes a severe V/Q mismatch and further causes hypoxemia. As a result of these factors, perfusion (CO) must be maintained and oxygenation (PaO_2, SaO_2, and CaO_2) restored for effective management of ARDS.

Patient Management

Management strategies for ARDS include correcting hypoxemia and acid-base disturbance, restoring cardiac function, and treating the underlying disease or other precipitating factors. This is generally accomplished with mechanical ventilation at 6 to 8 mL/kg of body weight and the application of PEEP to correct refractory hypoxemia. Patient tidal volume is based on an ideal body weight calculation. This is calculated from the following equation:

Male: $50 + 2.3\ (\text{Height in Inches} - 60) \times 6\ \text{mL}$
Female: $45.5 + 2.3\ (\text{Height in Inches} - 60) \times 6\ \text{mL}$

✓ Hypoxemia caused by intrapulmonary shunting is usually managed by mechanical ventilation with PEEP.

A high level of positive pressure may be required to produce adequate ventilation and oxygenation, but its adverse effect on cardiac function must be monitored carefully. Some patients may benefit from pressure-controlled ventilation (PCV) to limit the mean airway and alveolar pressures. In PCV mode, the inspiratory time is increased and pressures are generally reduced, but the mean airway pressure may remain nearly the same or slightly increased. This ultimately shortens the expiratory time, inverts the I:E ratio, and may potentially increase alveolar ventilation. As inspiratory time (T_I) increases and the elastic limit of the lung is reached, intrinsic PEEP increases the risk of air trapping and thus, may also increase the $PaCO_2$ while concomitantly increasing the occurrence of pneumothorax in an already stiff lung. These important adjuncts must be kept in fine balance, however, with each factor receiving equal consideration. To counter these trends, high frequency oscillation

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(HFO) is an important ventilator strategy when considering adjuncts for refractory hypoxemia and has been used with success at some facilities. (See references at the end of this case.)

✓ Pressure-controlled ventilation limits the inspiratory pressure during mechanical ventilation.

In an attempt to decrease the pressures exerted within the thorax, the patient was placed on pressure-controlled ventilation with an inverse ratio of 2:1 at pressure of 30 cm H_2O, T_I of 0.84 sec, F_IO_2 of 60%, and PEEP of 10 cm H_2O. Blood gases taken at the time revealed:

pH	7.43
$PaCO_2$	47 mm Hg
PaO_2	60 mm Hg
HCO_3^-	30 mEq/L
Hb	11.1 g %
SpO_2	90%
Mode	PC-IRV
T_I	0.84 sec
I:E ratio	2:1
f	14
PIP	30 cm H_2O
F_IO_2	60%
PEEP	10 cm H_2O

✓ The tidal volume delivered by pressure-controlled ventilation is directly related to the inspiratory pressure.

The pressures were then titrated in an effort to improve oxygenation and normalize her condition, but without success.

Key Medications

The patient was placed in a Rotorest® bed to help prevent the development of dependent atelectasis. She was also heavily sedated and maintained in a medicated coma for over 30 days but without significant improvement. She was given medication nebulizers with Proventil® every 4 hours for wheezing, lavaged with a combination of 2.0 mL normal saline (NS), 0.5 mL of 0.5% Proventil®, and 4.0 mL of 10% Mucomyst® solution, and suctioned prn with her treatment, to improve bronchopulmonary hygiene.

✓ Mucomyst and Pulmozyme are used to mobilize thick, retained secretions.

By this time she had a large amount of thick, yellow secretions. Dornase alpha (Pulmozyme®) (0.5 mL) and NS were administered via a small-volume nebulizer to loosen and remove the retained secretions.

Weaning

The deteriorating hemodynamic status prevented continuing use of inverse I:E ratio ventilation. She was returned to conventional ventilation throughout the remainder of her hospitalization. This included ventilation in the assist/control mode at a frequency of 24/min. The patient initiated inspiratory effort to 28/min. She was set to a V_T of 500 mL (approx. 9 mL/Kg), F_IO_2 of 80%, and PEEP of 8 cm H_2O. Arterial blood gases on these settings produced these results:

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pH	7.43
$PaCO_2$	48 mm Hg
PaO_2	64 mm Hg
HCO_3^-	31 mEq/L
Hb	11.1 g %
CaO_2	13.9 vol %
SpO_2	91%
Mode	A/C
f	24/min
V_T	500 mL
F_IO_2	80%
PEEP	8 cm H_2O

The frequency was increased to 30/min to override her excessive and demanding respiratory efforts. PEEP was increased to 10 cm H_2O, and F_IO_2 was reduced to 70%.

Blood gases were drawn 30 min later with the following results:

pH	7.51
$PaCO_2$	47 mm Hg
PaO_2	59 mm Hg
HCO_3^-	36.4 mEq/L
B.E.	12.4 mEq/L
Hb	9.6 g %
CaO_2	12.4 vol %
SpO_2	91%
Mode	A/C
f	30/min
V_T	500 mL
F_IO_2	70%
PEEP	10 cm H_2O

Several unsuccessful weaning attempts were made. Over several days she was weaned to a frequency of 24/min on assist/control (A/C) with a V_T of 380 mL (approx. 7 mL/Kg), F_IO_2 of 50%, and PEEP of 8 cm H_2O, and she remained on those settings for nearly one week. Due to her deteriorating condition and persistent complications, the family members and physician decided to remove her from ventilatory support, and she died a short time later.

✓ Low V_T is used to minimize the airway pressures.

Discussion

Through the course of hospitalization, the patient experienced many complicating injuries which contributed to her failure to recover. These included barotrauma to the lung parenchyma (pneumothorax was corrected with a chest tube), pneumomediastinum, and interstitial emphysema verified through serial chest radiographs, subcutaneous emphysema, and ultimately death. Many of these complications could have been expected as a result of her hospital course, because ARDS, even without her related complicating factors, carries a 50% mortality rate.

Data from a "low tidal volume" clinical trial showed approximately 25% fewer deaths among patients receiving small, rather than large, tidal volumes during mechanical ventilation. Medical centers have generally adopted this strategy as an evidence-based standard of practice for ARDS. Patient tidal volume is based on the ideal body weight calculation. This is calculated from the following equation:

Male: $50 + 2.3\ (\text{Height in Inches} - 60) \times 6\ \text{mL}$
Female: $45.5 + 2.3\ (\text{Height in Inches} - 60) \times 6\ \text{mL}$

Although the patient in this case study was not on this protocol, it seems worth mentioning that these guidelines have been proven to be useful in the management of ARDS. This is particularly true with the ARDSnet protocol for the treatment of acute lung injury (ALI) from any cause.

References (HFO and ARDS):

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Ackerman, M. H. (1993). The effect of saline lavage prior to suctioning, *Journal of Critical Care, 2*(4), 326–330.

Pedersen, C. R. (2009). Endotracheal suctioning of the adult intubated patient - what is the evidence? *Intensive and Critical Care Nursing, 25*(1), 21–30.

Reference (low tidal volume strategy):

The Acute Respiratory Distress Syndrome Network. (2000). Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *New England Journal of Medicine, 342*, 1301–1308.

CASE 10: MYASTHENIA GRAVIS

INTRODUCTION

G.L. was a 72-year-old Caucasian male (6'1", 74-Kg) with a history of myasthenia gravis for 25 years, but it had been in remission until about 6 months ago. His last hospitalization was 2 months ago because of ongoing shortness of breath, fatigue, inability to complete a sentence without taking a breath, chronic cough, malaise, yellow sputum production, and difficulty in swallowing. The diagnosis was pneumonia and he required mechanical ventilation for 17 days. He was eventually weaned off mechanical ventilation and discharged. During that hospitalization, he had repeated guaiac positive stool samples indicative of the presence of occult blood in the feces.

He had been home for 4 weeks and continued to have shortness of breath, fatigue, muscle weakness, orthopnea, and he had to sleep sitting up in his recliner. He came to the outpatient radiology clinic for barium and chest radiograph studies. Upon arrival to the radiology department he was extremely short of breath, and after his exams were completed he was referred to the emergency room. The patient declined to be seen and went home with his wife.

A barium study is done to evaluate the cause of guaiac positive stools.

A chest radiograph is done to evaluate the residual effects of pneumonia.

Within 5 min of arrival at home the patient suffered a respiratory arrest. His wife reported that he just stopped breathing and turned blue. The paramedics arrived and attempted to intubate but were unsuccessful after three attempts. While being transported to the ER, the patient had some spontaneous respirations but poor diaphragmatic movement.

The blood gas report indicates that the primary problem is acute ventilatory failure imposed on chronic ventilatory failure.

The initial blood gas analysis done upon arrival to the ER revealed the following results:

pH	6.95
$PaCO_2$	143 mm Hg
PaO_2	115 mm Hg
SaO_2	96%
HCO_3^-	30 mEq/L
Mode	Ambu bag and mask
F_IO_2	Estimated to be 100%
Notes	Poor ventilation

$PaCO_2$ indicates that the bag/mask system in the ambulance was not ventilating the patient.

HCO_3^- is elevated as a compensatory mechanism of the patient's preexisting ventilatory failure.

Indications

The patient was intubated in the ER and bagged at a frequency of 20/min and an F_IO_2 of 100%. Vital signs included: blood pressure 138/40 mm Hg, heart rate 60/min, temperature 95.2°F, SpO_2 97%, and Glasgow coma score 15. Bilateral breath sounds were present. A chest radiograph (Figure 19-6) showed proper ET tube position, good thoracic expansion, and barium particles in the left lower lobe.

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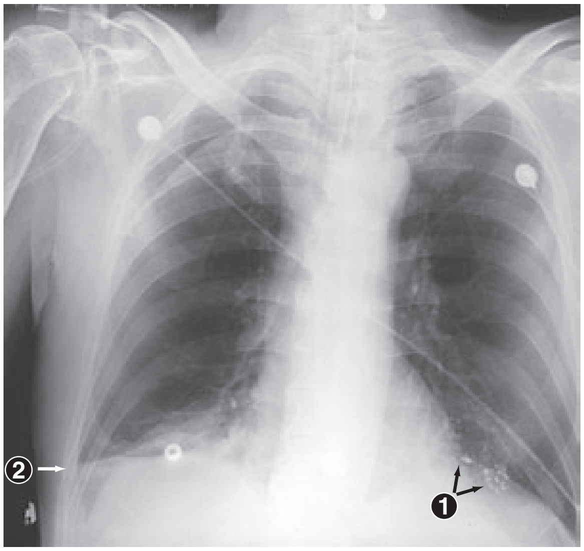

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FIGURE 19-6 Myathensia gravis. Aspiration of barium on the left side (1) is seen. The right hemidiaphragm is flat with a poorly defined costophrenic angle (2). This condition may be caused by pleural effusion.

Initial Settings

✓ Glasgow coma score of ≤8 indicates severe brain injury or neurological deficit. A score of 15 rules out presence of neurological deficit.

✓ The chest radiograph shows aspiration of barium in the left lower lobe.

✓ PaO_2 168 mm Hg is too high and the F_IO_2 should be decreased.

The patient was initially placed on volume-controlled ventilation with the following settings: CMV 16/min, V_T 750 mL, F_IO_2 80%, PEEP 0 cm H_2O. The patient had the following ABG results:

pH	7.47
$PaCO_2$	43 mm Hg
PaO_2	168 mm Hg
SaO_2	97%
HCO_3^-	30.5 mEq/L
Mode	CMV
f	16/min
V_T	750 mL
F_IO_2	80%
PEEP	0 cm H_2O

Patient Management

(Day 1) The patient was continued on mechanical ventilation and the F_IO_2 was weaned to 40%, with the following results:

✓ $PaCO_2$ 21 mm Hg is too low and the CMV frequency should be decreased.

pH	7.58
$PaCO_2$	21 mm Hg
PaO_2	122 mm Hg
SaO_2	96%
HCO_3^-	19.1 mEq/L
Mode	CMV

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f	16/min
V_T	750 mL
F_IO_2	40%
PEEP	0 cm H_2O

The patient's set frequency was decreased to 12/min and the blood gas results were:

pH	7.53
$PaCO_2$	25 mm Hg
PaO_2	128 mm Hg
SaO_2	97%
HCO_3^-	20 mEq/L
Mode	CMV
F	12
V_T	750 mL
F_IO_2	40%
PEEP	0 cm H_2O

$PaCO_2$ 25 mm Hg is still too low and the frequency should be decreased again.

The pressure support level should be titrated until a desired spontaneous tidal volume is obtained.

The CMV frequency was further decreased to 8/min for the rest of Day 1.

(Day 2) The next morning the patient was on CMV of 8/min, V_T 750 mL, F_IO_2 40%, and no PEEP. He had the following spontaneous parameters: V_E 9.7 L, V_T 660 mL, f 15/min, MIP −24 cm H_2O, and VC of 2,080 mL. The patient was alert and responsive. A CPAP/pressure support (CPAP/PS) trial was done with CPAP of 5 cm H_2O and PS of 5 cm H_2O, base flow of 2 L/min, and an F_IO_2 of 40%. The patient tolerated these settings for 4 hours with spontaneous tidal volumes of 600 to 700 mL and a spontaneous frequency of 16 to 21/min. Breath sounds showed fine inspiratory crackles throughout the anterior bases. All vital signs were within normal limits.

(Day 3) The patient was placed on CMV to rest overnight. Spontaneous parameters were: V_E 7.8 L, V_T 720 mL, frequency 12/min, MIP −12 cm H_2O, and a vital capacity of 4,020 mL. Heart rate, blood pressure, and temperature were all within normal limits. The patient's WBC spiked at a high of 16.2×10^3 (*normal 3.2 to 9.8* $\times$ 10^3), a chest radiograph showed the endotracheal tube was in proper position, and the bilateral opacities were consistent with barium aspiration.

The increased WBC may be due to aspiration of barium and subsequent aspiration pneumonitis.

He tolerated the previous CPAP/PS settings for 4 hours and was taken off CPAP/PS when he began to show extreme accessory muscle use. Blood gases during spontaneous breathing revealed:

pH	7.38
$PaCO_2$	34 mm Hg
PaO_2	87 mm Hg
SaO_2	97%
Mode	Spontaneous
F_IO_2	40%

$PaCO_2$ 34 mm Hg indicates hyperventilation. Prolonged hyperventilation may lead to fatigue of respiratory muscles.

(Day 4) The patient was rested overnight on CMV, and spontaneous parameters were: V_E 9.9 L, V_T 550 mL, f 18/min, MIP −26 cm H_2O, and VC of 3 L.

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Vital signs revealed: heart rate 76/min, temperature 38°C, blood pressure 120/74 mm Hg. The patient tolerated two trials of CPAP/PS at previous settings for 2 hours each time. Breath sounds were markedly decreased in both lower lobes. Patient was very anxious, restless, and had difficulty sleeping at night. He was started on Ativan for his anxiety. A feeding tube was ordered and placed for total nutrition.

(Day 5) He was rested overnight on CMV. Spontaneous parameters were: V_E 12.9 L, V_T 720 mL, f 18/min, MIP −29 cm H_2O, and VC of 3.4 L. Patient had a repeat endoscopy and was heavily sedated. A CPAP/PS trial was done before the patient was sedated. The spontaneous frequency was between 15/min and 17/min and the spontaneous tidal volume was in the low 400 mL range. Patient's vital signs were: heart rate 73/min, temperature 36.8°C, and blood pressure 120/59 mm Hg. The patient's endoscopy biopsy was positive for cytomegalovirus and he was started on Ganciclovir and Prilosec.

✓ Ganciclovir is an antiviral medication used to treat or prevent cytomegalovirus infections. Trade names are Cytovene and Cymevene.

(Day 6) The patient was again rested on CMV overnight. He was only able to tolerate partial spontaneous parameters and the results were: MIP −28 cm H_2O and VC of 1.1 L. The patient tolerated 90 min on CPAP/PS before he was extremely short of breath and exhibited an increased work of breathing with marked accessory muscle use. The patient's daily chest radiograph showed increasing bilateral infiltrates consistent with pneumonia/aspiration and the presence of barium in the left lower lobe. The patient had bilateral lower lobe rhonchi and was being suctioned for copious amounts of light-yellow secretions. The patient also spiked a temperature to 39°C. Pulmonary consultation was obtained at this time.

Patient Monitoring

The patient's spontaneous breathing parameters from Day 2 through Day 6 are summarized as follows. On Day 6, the patient could perform only part of the breathing parameters.

✓ The overall breathing parameters are not satisfactory for a weaning attempt.

Day	V_E (L)	V_T (mL)	f (/min)	MIP (cm H_2O)	VC (L)
2	9.7	660	15	−24	2.08
3	7.8	720	12	−12	4.02
4	9.9	550	18	−26	3.0
5	12.9	720	18	−29	3.4
6	N/A	N/A	N/A	−28	1.1

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Weaning

This patient was eventually weaned from mechanical ventilation after 25 days. He was weaned by using SIMV ventilation to build up his respiratory muscle strength before weaning to CPAP/PS again. The PS level was much higher than with previous attempts. The PS level was gradually weaned down each day as the patient's

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own spontaneous tidal volumes increased. He was then weaned to CPAP only and eventually extubated from CPAP to a face mask at 30% of oxygen.

A vigorous program of physical therapy was started to assist the patient with overall strengthening of his weakened muscle. A dietary consult was obtained and a metabolic study performed in order to adjust his feedings to appropriate nutritional levels needed for successful weaning.

The patient was also started on albuterol Q 4° with postural drainage and percussion. Antibiotics were used to treat the pneumonia. The patient was given neostigmine to manage his myasthenia gravis crisis.

NOTES: Adequate nutrition prior to and during weaning is necessary for a successful outcome. Appropriate nutrition helps to maintain and build respiratory muscle mass and strength, which enhances the likelihood of weaning.

✔ Neostigmine (Prostigmin) is an acetylcholinesterase inhibitor. It is used to improve muscle tone in people with myasthenia gravis and routinely in anesthesia at the end of an operation, to reverse the effects of nondepolarizing neuromuscular blockers such as rocuronium and vecuronium.

✔ Myasthenia gravis, aspiration pneumonia, and poor nutritional intake all contributed to the failure of earlier weaning attempts.

Complications

Weaning attempts on this patient were complicated by three major factors: (1) myasthenia gravis, (2) aspiration pneumonia, (3) premature weaning attempts.

The history of myasthenia gravis (a neuromuscular disease) inhibits the transmission of nerve impulses at the myoneural junction. These patients typically have chronic fatigability and weak muscles in the face and throat. They can also suffer what is known as a myasthenia gravis crisis—an acute exacerbation of muscle weakness that leads to respiratory distress and periods of apnea.

Aspiration pneumonia from the barium study increased the work of breathing and made it difficult for him to tolerate weaning procedures.

Premature and inconsistent weaning attempts during the first 3 days of his hospitalization made subsequent weaning attempts more difficult.

CASE 11: GUILLAIN-BARRÉ

INTRODUCTION

K.D. was a 14-year-old, 49-Kg, right-handed male who sustained two minor head injuries at school over a period of one week. One injury occurred while playing basketball when a heavier student fell on him, and the other occurred in an altercation during a break from school. Neither injury resulted in the loss of consciousness. He presented to the clinic complaining of headache and back pain but did not complain of slurred speech until being started on pain medication (Dolobid®) one day prior to admission. His medical history is noncontributory and consists of one hospitalization for a hernia repair in the distant past. He denied blurred vision but admitted to having transient nausea and had recently refused food. Since his slurred speech had become more pronounced than on his previous examination, he was admitted for further evaluation.

On examination, the patient was afibrile and had these vital signs: BP 110/70 mm Hg, pulse 78/min, respirations 14/min (up to 40/min on occasion). His facial muscles showed symmetry bilaterally. He had trouble sitting up due to excessive pain in his lower extremities, especially on the left. Lung sounds revealed basilar crackles on the right with decreased aeration on the left. A chest radiograph showed left lower lobe atelectasis. The cranial nerves 2 to 12 appeared intact but motor strength showed marked weakness. The patient was able to lift his right heel 2 to 3 inches with great difficulty but was unable to lift his left heel at all. His deep tendon reflexes were completely absent.

Laboratory results revealed WBCs of 14,000 with 84% segs. The electrolytes and urinalysis were within normal limits. His FEV_1 and FVC were 1.4 L and 1.7 L, respectively. The FEV_1/FVC ratio was 82%. A CT scan of the brain revealed a completely normal study. A chest radiograph at the time showed bilateral consolidation secondary to muscle weakness and left lower lobe atelectasis.

✓ Restrictive lung disease typically shows reduction in FEV_1 *and* FVC, resulting in a normal FEV_1/FVC%.

Indications

The patient had a 5-day history of progressive muscular weakness. For this reason, basic spirometry was done Q4 hours through the night. His predicted forced vital capacity (FVC) was 3.38 L and his initial vital capacity was 1.7 L (50% of the predicted value), which he maintained through the early evening. His predicted FEV_1 was 3.12 L and his initial measurement revealed 1.4 L (48% of predicted) for an 82% FEV_1/FVC ratio. By 3:30 a.m., the patient's vital capacity had dropped to 1.2 L (35% of predicted) and an arterial blood gas (ABG) at that time, on room air, revealed the following:

✓ Mechanical ventilation is indicated when the vital capacity falls below 1,000 mL or twice the patient's predicted tidal volume.

pH	7.37
$PaCO_2$	45 mm Hg
PaO_2	46 mm Hg
SaO_2	76%
HCO_3^-	24.9 mEq/L
f	24/min
F_IO_2	21%

Oxygen therapy was initiated at 2 L/min per nasal cannula. He rested well until approximately 8:20 the next morning. The vital capacity at that time was slightly over 1 L (30% of predicted) and the ABGs revealed:

pH	7.39
$PaCO_2$	43 mm Hg
PaO_2	52 mm Hg
SaO_2	86%
HCO_3^-	25.2 mEq/L
f	22/min

Every attempt was made to forestall clinical deterioration. The trends of bedside spirometry and serial blood gases showed that the patient was progressing

✓ Bilateral breath sounds suggests proper endotracheal intubation. This sign should coincide with absence of respiratory distress, presence of adequate SpO_2, and stable vital signs.

to ventilatory failure. By 10:30 a.m., the patient's vital capacity had dropped to 0.7 L (20% of predicted) and maintaining adequate oxygenation was becoming more difficult. Consequently, a decision was made to perform elective intubation. Family members were informed of the decision and the respiratory therapist gathered the necessary equipment for a controlled intubation. The patient was mildly sedated and orally intubated with a size 7.5 ET tube without difficulty. Bilateral breath sounds were heard and a portable chest radiograph was ordered.

Initial Settings

Following intubation and confirmation of proper tube placement, the patient was placed on volume-controlled ventilation in assist/control mode (A/C), backup frequency of 12, tidal volume of 600 mL (approx. 12 mL/Kg), and F_IO_2 of 40%. PEEP was not initiated at that time. ABGs revealed the following:

pH	7.36
$PaCO_2$	43 mm Hg
PaO_2	53 mm Hg
SaO_2	86%
HCO_3^-	23.7 mEq/L
Mode	A/C
f	12/min
Spont f	17 min
V_T	500 mL
F_IO_2	40%

The ventilator settings were changed accordingly to a backup frequency of 14/min, F_IO_2 of 50%, and PEEP of 5 cm H_2O. Follow-up ABGs showed:

pH	7.45
$PaCO_2$	42 mm Hg
PaO_2	73 mm Hg
SaO_2	94%
HCO_3^-	28 mEq/L
Mode	A/C
f	14/min
V_T	500 mL
F_IO_2	50%
PEEP	5 cm H_2O

The patient remained relatively stable and was monitored closely for signs of respiratory distress, difficulty breathing, or ventilator dyssynchrony. The ventilator peak flow was adjusted to meet the patient's inspiratory demand without compromise to alveolar ventilation. By the third ventilator day, the F_IO_2 was increased to 60% and the PEEP was increased to 8 cm H_2O. These changes were made to prevent atelectasis and improve alveolar ventilation as evidenced by a declining PaO_2 on his morning blood gas. These revealed the following:

pH	7.43
$PaCO_2$	35 mm Hg
PaO_2	57 mm Hg
SaO_2	87%
HCO_3^-	22.5 mEq/L
Mode	A/C
f	14/min
V_T	500 mL
F_IO_2	60%
PEEP	8 cm H_2O

The patient's PEEP was subsequently increased to 10 cm H_2O without cardiac or hemodynamic compromise. F_IO_2 was decreased to 50% because of improving SpO_2 values. Follow-up ABGs revealed:

pH	7.45
$PaCO_2$	35 mm Hg
PaO_2	132 mm Hg
SaO_2	97%
HCO_3^-	23.5 mEq/L
Mode	A/C
f	14/min
V_T	500 mL
F_IO_2	50%
PEEP	10 cm H_2O

The F_IO_2 was again lowered to 40% and he remained stable for the next 2 days.

Key Medications

On day 4 of mechanical ventilation, a persistent low-grade fever and thick tenacious secretions necessitated initiation of vigorous pulmonary toilet. Albuterol with Mucomyst via a small-volume nebulizer was administered Q4 hours for wheezing given inline with the ventilator circuit. Postural drainage and percussion were done to the lower lobes. Bronchoalveolar lavage with 4 mL of 10% Mucomyst, 0.5 mL of 0.5% albuterol, and 5 mL of normal saline facilitated suction of moderate to large amounts of thick secretions

Gradual clearing of retained secretions and atelectasis became evident by follow-up chest radiographs. These management strategies in pulmonary toilet were continued for the remainder of the ventilatory period.

Immobility was a major factor in the patient's clinical deterioration. Extensive physical therapy was begun with range of motion and activity as tolerated. However, extreme pain made these sessions almost unbearable to the patient.

Each week of muscle immobility requires 4 weeks of rehabilitation of his diminished muscle mass.

Patient Monitoring

Respiratory parameters were obtained every morning in an effort to trend his ventilatory mechanics. The parameters listed in Table 19-1 were obtained on representative days and are not necessarily concurrent (NOTE: on ventilator days 6 through 25 the patient was mainly unable to breathe on his own).

Patient Management

Management strategy for Guillain-Barré was mainly supportive, since the ascending paralysis typically runs its course. Because this disease pathology presents signs and

TABLE 19-1 Respiratory Parameters

Vent Day	f (/min)	V_T (mL)	FVC (L)	V_E (L)	f/V_T	MIP (cm H_2O)	Compliance (mL/cm H_2O)
2	37	140	0.67	5.30	264	−20	28
3	50	60	0.32	5.30	833	−15	32
4	25	80	0.69	2.60	312	−13	38
5	23	80	0.10	5.40	287	−2	62
26	6	410	0.41	4.90	15	−15	45
28	10	440	0.45	5.00	23	−15	52
32	23	70	0.65	1.03	328	−10	34
36	30	135	0.55	4.00	222	−26	48
42	29	110	0.54	3.10	263	−11	41
46	32	85	0.25	2.73	376	−10	44
52	27	200	0.36	4.20	135	−14	51
54	23	210	0.57	4.83	1.09	−17	53
56	32	115	0.52	3.70	278	−17	55
58	21	240	0.68	4.60	88	−15	64
60	25	250	0.96	6.22	100	−23	70
62	29	175	1.02	5.09	165	−24	58
64	25	240	0.68	5.89	104	−26	55
66	19	210	1.15	4.91	90	−30	54
68	18	200	1.07	6.11	90	−37	54

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symptoms that are predominantly idiopathic in nature, treatment included plasmaphoresis in an attempt to reverse the pathology. However, by the sixth ventilator day it was apparent that long-term management strategies would be necessary. As such, a pediatric feeding tube was inserted and a tracheotomy was performed in an effort to facilitate removal of secretions and to make oral care more accessible. A metabolic study was performed to ascertain the patient's nutritional status, and feedings were adjusted in terms of optimizing the resulting V/Q ratio and total caloric intake. He was placed in a Rotorest® bed and continuously turned from side to side and kept comfortable with anti-anxiolytics. The patient was closely monitored for signs of hypercapnea, hypoxia, and respiratory distress. It is important to note that the patient was not able to initiate any significant spontaneous tidal volume. The ventilator was thus completely responsible for his total alveolar ventilation. Blood gas analysis after the tracheostomy by general anesthesia revealed the following:

✓ Tracheotomy is performed when long-term mechanical ventilation is anticipated.

pH	7.40
$PaCO_2$	52 mm Hg
PaO_2	122 mm Hg
SaO_2	96%
HCO_3^-	31 mEq/L
Mode	A/C
f	14/min
V_T	500 mL
F_IO_2	40%
PEEP	10 cm H_2O

In order to improve alveolar ventilation, the frequency was increased to 16/min and the tidal volume was increased to 670 mL (approx. 13 mL/Kg) because he was still unable to initiate spontaneous ventilation but appeared "air-hungry" (dyspnic). Due to his unstable ventilatory status, blood gas results from the following morning revealed:

pH	7.39
$PaCO_2$	48 mm Hg
PaO_2	90 mm Hg
SaO_2	94%
HCO_3^-	28 mEq/L
Mode	A/C
f	16/min
V_T	670 mL
F_IO_2	40%
PEEP	10 cm H_2O

In order to minimize airway pressures and reduce the risk of barotrauma, the patient's $PaCO_2$ was maintained in the mid to upper 40s (permissive hypercapnea). This is an appropriate ventilation strategy as long as the physician and health care team members "buy-in" to the stated goals for mechanical ventilation for a given pathology. The patient appeared to rest comfortably. Blood gases obtained on the 18th ventilator day revealed:

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pH	7.48
$PaCO_2$	47 mm Hg
PaO_2	114 mm Hg
SaO_2	97%
HCO_3^-	34 mEq/L
Mode	A/C
f	18/min
V_T	670 mL
F_IO_2	40%
PEEP	10 cm H_2O

These parameters were maintained until the 24th ventilator day, when the patient appeared to improve clinically and began to breathe significantly on his own. The patient became increasingly uneasy about his breathlessness. As a result, the settings were changed to SIMV mode, frequency of 18/min, tidal volume of 700 mL (approx. 14 mL/Kg), and F_IO_2 of 40%. PEEP was reduced to 5 cm H_2O and pressure support was initiated to augment his spontaneous volume. Blood gases taken after these changes indicated marked improvement in oxygenation and acid-base balance:

pH	7.47
$PaCO_2$	38 mm Hg
PaO_2	117 mm Hg
SaO_2	94%
HCO_3^-	27 mEq/L
Mode	SIMV
f	18/min
V_T	700 mL
F_IO_2	40%
PEEP	5 cm H_2O
PS	15 cm H_2O

At this point, the F_IO_2 was reduced to 35% and remained relatively stable for the next 19 days. A bronchoscopy was performed on ventilator day 32 to clear an obstructed left lower lobe bronchus. This was achieved without difficulty and, by ventilator day 39, the frequency was decreased from 18 to 12/min. The following day, the frequency was decreased to 10/min. His spontaneous frequency ranged between 29 and 32/min.

Weaning

On ventilator day 40, the patient continued to show evidence of improved alveolar ventilation. Brief T-piece (Briggs "T" adaptor) trials off the ventilator, sometimes as little as 10 to 15 minutes every 2 to 4 hours, were initiated to encourage the use of respiratory muscles. Gradually, by the 54th ventilator day, he was able to tolerate short periods off the ventilator, from 30 minutes to an hour twice a day, and on CPAP with minimal pressure support (10 to 15 cm H_2O) the remainder of the

✓ The pressure support level is titrated and subsequently adjusted until spontaneous tidal volume = 10 to 15 mL/kg or spontaneous frequency <25/min.

day. At this pressure support range, the patient was able to achieve a tidal volume of 300 to 400 mL. As muscle strength returned and gradually improved, less pressure support was necessary to maintain an adequate tidal volume. The patient was allowed to rest completely at night on A/C mode, and weaning was resumed each morning to CPAP with low level of pressure support.

By day 59, the patient was able to tolerate 40 minutes to an hour off the ventilator every 2 to 4 hours during the day while resting at night. Each day brought the same monotonous routine, but the patient continued to improve with longer periods free from the ventilator. On ventilator day 68 he was able to remain off the ventilator 24 hours per day. Rehabilitation had just begun!

Complications

This patient encountered almost complete paralysis in the acute pathologic phase of his disease as he was only able to blink his eyes. He was under severe emotional and psychological stress throughout his hospitalization due to his complete paralysis and his reliance on others for care. He developed GI bleeding from a duodenal ulcer for which he was treated with Zantac® via his feeding tube. Otitis media developed and was treated with broad-spectrum antibiotics, and a persistent left lower lobe pneumonitis required bronchoscopy. Rehabilitation was begun and he was transferred out of the intensive care unit with humidified O_2 at an F_IO_2 of 30%. The tracheostomy was gradually buttoned and eventually removed. He required only 2 L/min of oxygen via nasal cannula while recovering in rehab. The patient was discharged after 154 days of hospital stay (last 94 days in extensive rehabilitation) on room air requiring only 25 mg of prednisone and follow-up physical therapy. He continued to progress and, eventually, demonstrated no residual effects from this debilitating illness.

References (lavage and suctioning):

Ackerman, M. H. (1993). The effect of saline lavage prior to suctioning, *Journal of Critical Care*, *2*(4), 326–330.

Pedersen, C. R. (2009). Endotracheal suctioning of the adult intubated patient—what is the evidence? *Intensive and Critical Care Nursing*, *25*(1), 21–30.

CASE 12: BOTULISM

INTRODUCTION

J.D. was a 66-year-old, 50-Kg female in her usual state of health until she consumed a partial jar of home-canned salsa. The following day she developed abdominal cramping thought to be the flu. Her intermittent cramping continued for 48 hours post-ingestion, and she subsequently developed slurred speech and blurred vision.

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In addition to these symptoms, the patient experienced vertigo and diplopia at times and sought medical attention when diffuse upper motor weakness appeared. Her bulbar weakness and generalized fatigue necessitated admission for close observation in the ICU. *Clostridium botulism* was cultured from the salsa and also found to be present in the patient's stool. Community and state health officials were contacted for an antitoxin, and subsequently this case was reported to the Centers for Disease Control and Prevention (CDCP) in Atlanta. Within 24 hours of admission the patient received trivalent botulism antitoxin, but she encountered progressive deterioration with concomitant respiratory compromise.

Indications

On examination, the patient exhibited no evidence of distress. She did, however, manifest bilateral ptosis (drooping of eyelids) with decreased facial expression. Her neck was supple, lungs were clear, and her mental status appeared normal. Pupillary light reflexes revealed sluggish reactions bilaterally. The tongue was midline with evidence of slurred speech. Motor examination revealed diffuse weakness of the upper extremities. Symmetrical fatigability of the biceps, deltoids, and grips was also noted. Gait revealed mild weakness, and electromyogram (EMG) studies showed decreased amplitude of repetitive nerve action potentials.

The patient was admitted for close observation. Vital capacities and maximum inspiratory pressure (MIP) were done Q2 hours to assess her ventilatory status. Her white blood cell count was 6,900 on admission and 12,500 (*normal 3.2 to 9.8* $\times$ *10^3*) 48 hours later. Vital signs revealed blood pressure of 168/56 mm Hg, heart rate of 88/min, temperature of 36.4°C, and respirations of 24/min. Her initial arterial blood gases on room air revealed:

pH	7.46
$PaCO_2$	36 mm Hg
PaO_2	72 mm Hg
SaO_2	95%
HCO_3^-	24.5 mEq/L
f	24/min
F_IO_2	21%

Her vital capacity at 03:30 was 1.6 L (62% of predicted) with a MIP of -18 cm H_2O. By 08:00, the condition of the patient remained relatively unchanged except that her MIP had decreased to -12 cm H_2O. Due to progressive fatigue of respiratory muscles, by 10:00, she was nasally intubated with a size 7.5 ET tube to facilitate mechanical ventilation.

In spite of normal $PaCO_2$, mechanical ventilation is indicated because of her progressive muscle weakness (decreasing MIP), impending fatigue, and anticipated progression of botulism poisoning.

Initial Settings

She was started on volume-controlled ventilation in assist/control (A/C) mode, backup frequency of 12/min, tidal volume of 400 mL (approx. 8 mL/Kg), PEEP of 5 cm H_2O, and an F_IO_2 of 35%. ABGs drawn 30 min later revealed the following:

pH	7.55
$PaCO_2$	25 mm Hg
PaO_2	120 mm Hg
SaO_2	96%
HCO_3^-	21.1 mEq/L
Mode	A/C
f	12/min
Spont f	22/min
V_T	400 mL
F_IO_2	35%
PEEP	5 cm H_2O

Based on these results, the frequency was decreased to 8/min and the F_IO_2 was reduced to 30%. ABGs were ordered for the following morning. The results showed:

pH	7.43
$PaCO_2$	37 mm Hg
PaO_2	110 mm Hg
SaO_2	97%
HCO_3^-	23.6 mEq/L
Mode	A/C
f	8/min
Spont f	12/min
V_T	400 mL
F_IO_2	30%
f	5 cm H_2O

The patient remained febrile and weak as manifested by serial MIPs. On the 5th ventilator day, the patient underwent an elective tracheostomy for comfort and to prevent development of septic sinusitis from prolonged nasal intubation. She tolerated the procedure well.

Patient Monitoring

Her respiratory mechanics showed no signs of improvement through the first week to 10 days but gradually improved over time. She was monitored for progressive muscle strength with serial forced vital capacities (FVC) and maximum inspiratory pressure (MIP) measurements while on CPAP every morning, which indicated gradual improvement. The serial measurements are listed in Table 19-2. The patient

TABLE 19-2 Serial Vital Capacities and Maximum Inspiratory Pressure

Vent Day	f (/min)	FVC (L)	MIP (cm H_2O)
1	18	1.03	−30
2	22	1.30	−30
3	28	1.11	−33
4	32	1.13	−36
5	34	1.06	−35
6	30	1.45	−39
7	28	1.42	−36
8	28	1.21	−50
9	24	1.40	−38
10	20	1.80	−34
11	24	1.70	−38
12	24	1.60	−40
13	18	1.65	−43
14	18	2.10	−46

continued to improve over the latter half of her hospitalization, and after 14 days of mechanical ventilation she exhibited evidence that she could support her own respiratory status without assistance from the ventilator.

Patient Management

✓ The management strategy for botulism consists mainly of supportive measures.

The management strategy for botulism consists mainly of supportive measures, as she was mechanically ventilated for 14 days. Patient supportive measures included turning the patient from side to side, assisting the patient to an Orthochair at the bedside, and using medications for comfort. Her nutritional status was maintained through I.V. fluids alone. The patient continued to improve, and on the 7th ventilator day she was transferred to a subacute unit for further recovery and monitoring.

Key Medications

The patient's lungs remained diminished but clear throughout. There were no abnormal secretions when she was suctioned. However, vigorous pulmonary toilet was done to prevent lung pathology while in the step-down ICU. She was treated, prophylactically, on small-volume nebulizer therapy with 0.5 mL of 0.5% albuterol

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in 2.0 mL of saline Q 4 hours for wheezing, inline with the ventilator circuit. This medication was discontinued after her removal from the ventilator.

Weaning

Beginning on the 10th day of mechanical ventilation, the patient began CPAP trials. She tolerated this procedure well for up to 8 to 10 hours at a time. She was then returned to SIMV mode with a backup frequency of 6/min, tidal volume of 400 mL (approx. 8 mL/Kg), PEEP of 5 cm H_2O, F_IO_2 of 30%, and pressure support (PS) of 10 cm H_2O to rest for the night. Her frequency remained in the mid to high 20s and her return volume was consistently between 350 mL and 450 mL. She did not complain of shortness of breath, difficulty of breathing, or chest discomfort, and appeared to rest comfortably. By the 12th day of continuous mechanical ventilation, the patient was able to tolerate CPAP of 5 cm H_2O and F_IO_2 of 30% 24 hours/day. Her ABGs revealed the following:

pH	7.44
$PaCO_2$	46 mm Hg
PaO_2	91 mm Hg
SaO_2	96%
HCO_3^-	29.9 mEq/L
Mode	CPAP
Spont f	28/min
F_IO_2	30%
PEEP	5 cm H_2O

At that point, she was removed from the ventilator and placed on a humidified T-piece (Briggs "T" adaptor) at an F_IO_2 of 30%. She was monitored via a continuous-pulse oximeter that revealed SpO_2 between 92% and 94%. Her frequency remained in the low 20s. She was closely observed for signs of respiratory distress or tachypnea (>30/min). Three days later, ABGs again were obtained while on the T- piece at an F_IO_2 of 30%. These revealed:

pH	7.46
$PaCO_2$	45 mm Hg
PaO_2	87 mm Hg
SaO_2	96%
HCO_3^-	30.9 mEq/L
f	24/min

On the 14th day, her tracheostomy was buttoned and she was placed on a nasal cannula at 2 L/min without complications. She was decannulated two days later.

Complications

The patient showed no serious and lasting complications from this episode of botulism and was discharged from the hospital on room air 21 days following her admission. She did exhibit some generalized weakness and mild ptosis that continued to improve after discharge.

CASE 13: MECONIUM ASPIRATION/ PATENT DUCTUS ARTERIOSUS

INTRODUCTION

Long gestational age has a higher incidence of meconium aspiration syndrome.

Visualization of the vocal cords helps to determine if meconium is present in the upper airway. If the vocal cords are stained green, suction should be done prior to the first breath so as to prevent or minimize meconium aspiration.

A 22-year-old Caucasian primigravida mother gave birth to a 41-week-gestation female. The pregnancy was uncomplicated and the mother received all scheduled prenatal care. Following admission to the labor and delivery floor, the amniotic membrane was ruptured, revealing grade III meconium-stained amniotic fluid. Due to failure to progress past 6-cm dilation, a cesarean section was performed. The birth weight was 3,840-gm (8.5 lb).

At delivery, the neonate presented as nonvigorous. There was no respiratory effort, a heart rate of 75 beats/min, poor peripheral perfusion, and poor muscle tone. The vocal cords, stained green from meconium, were visualized, and the neonate was intubated immediately with a size 3.5 endotracheal (ET) tube and suctioned via meconium aspirator attached to the ET tube. A large amount of particulate meconium was recovered. A second attempt at suctioning via the ET tube recovered a small amount of meconium. Following the second intubation and suction, the neonate initiated her first breath. Respirations were labored with the presence of mild intercostal and substernal retractions, nasal flaring, and audible grunting.

Points for Discussion

1. What would be the next step in providing support to this neonate?
2. What are your concerns regarding her postresuscitative care?

The Apgar scores were 3 and 7 at 1 and 5 mins, respectively. They were scored as shown below.

	0	**1**	**2**	**1 min**	**5 min**
Heart rate	None	Slow Irregular	Over 100	1	2
Respiratory effort	Apnea	Irregular shallow Gasping	Yelling, crying	0	1
Muscle tone	Flaccid	Some flexion of extremities	Well-flexed	1	2
Reflex	No response to stimulus	Grimace	Crying	0	1
Color	Pale blue	Blue extremities	Pink all over Body pink	1	1
Total				3	7

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Indications

Continuous positive airway pressure (CPAP) was administered via a T-piece resuscitator to support the infant's respiratory effort. After stabilization in the delivery room, the infant was admitted to newborn intensive care unit (NICU) for further observation, evaluation, and treatment with antibiotics. The infant was placed on a heated/humidified head box at 40% oxygen. Within a few hours after admission, she became dusky and presented with moderate to severe intercostal and substernal retractions. As her condition continued to deteriorate, an umbilical artery catheter (UAC) and an orogastric (OG) tube were placed, and CPAP at 5 cm H_20 via nasal prongs on an F_IO_2 of 60% was initiated.

pH	7.28
$PaCO_2$	48 mm Hg
PaO_2	40 mm Hg
HCO_3^-	22 mEq/L
B.E.	−2 mEq/L
Hct	41%
Mode	CPAP at 5 cm H_20 via nasal prongs
F_IO_2	60%

In spite of CPAP and 60% oxygen, the infant's oxygenation status continued to deteriorate. Her respiratory frequency increased as she tried to compensate for the hypoxia. Capillary refill time was increased from 4 to 5 sec. Her extremities were cold and the mean arterial pressure was 35 mm Hg.

Based on the condition and prognosis of this infant, she was electively intubated with a 3.5 mm endotracheal (ET) tube and placed on SIMV via a time-cycled pressure-limited ventilator.

✓ Increased capillary refill time indicates shunting of blood flow from the extremities or increased peripheral vascular resistance.

Initial Settings

The predicted tidal volume (V_T) was estimated by using a 4- to 6 mL/Kg range. At a weight of 3.8Kg, the estimated V_T range is 15.2–22.8 mL. The initial settings of the time-cycled pressure-limited ventilator were: SIMV, f 40/min, PIP/PEEP 24/5 cm H_2O, T_{INSP} 0.5 sec, and F_IO_2 100%. At these settings, the mean airway pressure (mPaw) was 14.5 cm H_2O, V_T was 13.3 mL, and blood gases from a UAC sample showed:

pH	7.32
$PaCO_2$	44 mm Hg
PaO_2	43 mm Hg
HCO_3^-	22.2 mEq/L
B.E.	−2.9 mEq/L
Mode	SIMV
PIP/PEEP	24/5 cm H_2O
f	40/min
T_{INSP}	0.5 sec

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PIP of 24 cm H_2O should be increased to 28 cm H_2O to achieve the estimated V_T, which may enhance alveolar recruitment and oxygenation.

F_IO_2	100%
mPaw	14.5 cm H_2O

The PIP/PEEP were changed to 28/6 cm H_2O and the f was changed to 50/min. At this PIP, the V_T has increased to 15.5mL. Also, blood gases and transcutaneous gas tension showed:

Since the initial f is 40/min (or 1.5 sec cycle time) and the T_{INSP} is 0.5 sec, the T_{EXP} is 1.5 − T_{INSP} = 1.5 − 0.5 sec = 1 sec. The I:E ratio is therefore 1:2.

pH	7.41
$PaCO_2$	34 mm Hg
PaO_2	45 mm Hg
HCO_3^-	21.1 mEq/L
B.E.	−2.7 mEq/L
SpO_2	86%
$P_{tc}O_2$	28 mm Hg
$P_{tc}CO_2$	30 mm Hg
Mode	SIMV
PIP/PEEP	28/6 cm H_2O
f	40/min
T_{INSP}	0.5 sec
F_IO_2	100%
mPaw	16.5 cm H_2O

The increase in PIP and PEEP causes a higher mPaw (from 14.5 to 16.5 cm H_2O). This may (1) lower the production of natural surfactant, (2) cause patent ductus arteriosus (PDA), and (3) increase the risk of barotrauma. Pre- and postductal saturations were measured, revealing a preductal SpO_2 of 86% and a postductal SpO_2 of 70%.

An echocardiogram was performed at this time, and it confirmed a patent ductus arteriosus (PDA) with a right-to-left shunt and evidence of persistent pulmonary hypertension of the newborn. The patient was started on 0.2 mg/kg of indomethacin. Administration of indomethacin includes 3 doses, with the second being 12 hours after the first, and the third dose 24 hours after the second.

Twelve hours after the second indomethacin administration, the blood gases showed:

pH	7.59
$PaCO_2$	27 mm Hg
PaO_2	66 mm Hg
HCO_3^-	24.9 mEq/L
B.E.	3.1 mEq/L
SpO_2	96%
$P_{tc}O_2$	53 mm Hg
$P_{tc}CO_2$	28 mm Hg
Mode	SIMV
PIP/PEEP	28/6 cm H_2O
f	40/min
T_{INSP}	0.3 sec
F_IO_2	100%
mPaw	16.5 cm H_2O

The PDA has partially closed allowing a decrease of the right-to-left shunt. This lowers the peak inspiratory pressure and improves gas exchange. The risk of barotrauma is therefore lowered.

Over the course of the next 12 hours, the infant slowly began to wean from the ventilator. The following results were obtained:

pH	7.38
$PaCO_2$	42 mm Hg
PaO_2	56 mm Hg

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✓ The administration of surfactant helps to increase the pulmonary compliance and to lower the peak inspiratory pressure, F_IO_2, and mPaw requirements. Although most often used in infants <34 weeks, it may be considered in term infants to improve compliance.

✓ An important aspect of weaning in this case is to maintain the PaO_2 at a safe level (50s mm Hg), to prevent increase of PVR and reopening of PDA.

HCO_3^-	24.2 mEq/L
B.E.	−0.9 mEq/L
SpO_2	90%
$P_{tc}O_2$	47 mm Hg
$P_{tc}CO_2$	66 mm Hg
Mode	SIMV
PIP/PEEP	25/6 cm H_2O
f	30/min
T_{INSP}	0.5 sec
F_IO_2	50%
mPaw	12 cm H_2O

Over the next 2 days the ventilator was weaned by gradually decreasing the PIP, PEEP, f, and F_IO_2. The patient continued to improve and was finally extubated to an oxyhood at 30% F_IO_2.

Key Medications

Medication	Main Purpose
Decadron	Improves lung function in infants requiring prolonged ventilation. It also decreases tracheal edema to facilitate extubation
Indomethacin	Used for symptomatic treatment of PDA in infants when the ductus arteriosus fails to close on its own. Complete closure usually results within 72 hours of birth.
Albumin	Used for volume expansion when treating RDS. Shock may be seen after correction of acidosis or hypoxia (due to ↓ systemic vascular resistance and ↓ blood pressure)
Ampicillin	Broad-spectrum antibiotic
Surfactant	Treatment of RDS by increasing the lung compliance and decreasing the alveolar surface tension

Special Considerations

✓ Meconium aspiration is most likely to occur in term or postterm infants who have intrauterine distress or hypoxia.

Meconium aspiration is most likely to occur in term or postterm infants who have intrauterine distress or hypoxia. When meconium is seen in the amniotic fluid, and if the infant presents as nonvigorous (as outlined by NRP guidelines), they should be orally intubated and suctioned via a meconium aspirator. This is done to prevent aspiration of meconium and airway obstruction. If the infant has taken initial breaths following delivery and presents as vigorous, endotracheal intubation is ***not*** indicated.

An infant diagnosed with meconium aspiration runs a high risk of barotrauma when mechanical ventilation is instituted. Ongoing assessment of the chest is of vital importance. Monitoring the mPaw is the best indicator of impending barotraumatic events. It is important to keep the mPaw as low as possible.

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Insufficient PaO_2 may cause hypoxemia and an increase of the PVR.

With the presence of PDA revealed by the echocardiogram, an adequate PaO_2 should be provided and maintained in an attempt to minimize an increase of the pulmonary vascular resistance (PVR). Attempts to reduce the F_IO_2 requirement may be successful, but this must be done slowly to prevent the PVR from increasing.

CASE 14: PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN

INTRODUCTION

The 5-min Apgar score should show significant improvement over the one-minute score.

A 16-year-old primigravida mother gave birth to a 28-week-gestation, 1,130-gm male infant born without prenatal care. The 1- and 5-min Apgar scores were 3 and 5, respectively.

	0	**1**	**2**	**1 min**	**5 min**
Heart rate	None	Slow Irregular	Over 100	1	1
Respiratory effort	Apnea	Irregular shallow Gasping	Yelling, crying	1	1
Muscle tone	Flaccid	Some flexion of extremities	Well-flexed	0	1
Reflex	No response to stimulus	Grimace	Crying	0	1
Color	Pale blue	Blue extremities	Pink all over Body pink	1	1
Total				3	5

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Peripheral perfusion status is a gross indicator of cardiac output or tissue perfusion.

Despite an Apgar score of 5 at 5 min, the cardiopulmonary status of the infant continued to deteriorate and cardiopulmonary resuscitation (CPR) became necessary. CPR was started using 100% oxygen via a flow-inflating resuscitation bag with pressure manometer attached. The infant was subsequently intubated with a 2.5 mm endotracheal tube, stabilized and placed on pressure-controlled ventilation without further complications.

An umbilical artery catheter (UAC) was inserted and secured. After a period of stabilization on the ventilator, the infant still appeared dusky. His capillary refill

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was slow, indicating poor peripheral perfusion, but the extremities were warm. The anterior fontanel was soft with slightly overlapping sutures. Breath sounds were diminished bilaterally.

The blood gases show oxygenation failure because the PaO_2 is far below normal and the $PaCO_2$ of 48 mm Hg is only slightly above normal.

Arterial blood was drawn from the UAC while on the ventilator with a set frequency of 40/min, PIP/PEEP of 28/4 cm H_2O, and 100% oxygen. The blood gas results were:

UAC Sample	
pH	7.28
$PaCO_2$	48 mm Hg
PaO_2	22 mm Hg
HCO_3^-	21.6 mEq/L
B.E.	−7.2 mEq/L
Mode	SIMV
F_IO_2	100%
PIP	28 mm Hg
PEEP	4 cm H_2O
f	40/min

Several tests are available to confirm PPHN, including physical exam, lability of oxygenation, preductal and postductal oxygenation saturation measurements (5% difference—PDA presents with a greater difference in pre-/postductal oxygen saturations), chest radiograph, and color Doppler echocardiography.

Doppler echocardiography confirmed the presence of PPHN as indicated by the pre- and postductal saturation measurements.

During a 24-hour period, unsuccessful attempts were made to decrease the oxygen requirement. Very little progress was seen in the patient as the pulmonary hypertension was not resolved using the hyperventilation strategy. Inhaled nitric oxide (iNO) therapy was started in the hope of reversing the pulmonary hypertension.

The patient received 55 ppm (parts per million) of iNO, and the blood gases after 1 hour of iNO therapy showed:

UAC Sample	
pH	7.45
$PaCO_2$	35 mm Hg
PaO_2	105 mm Hg
Mode	SIMV
PIP/PEEP	32/6 cm H_2O
f	45/min
T_{INSP}	0.24 sec
F_IO_2	85%

The infant's F_IO_2 requirement continued to decrease over the next 48 hours. iNO therapy was discontinued and the F_IO_2 was weaned to 45%. Conventional ventilation continued at a frequency of 45/min.

Patient Management

The pulmonary vascular resistance of the infant was increased due to severe hypoxemia and hypoxic vasoconstriction. Since hypoxemia could not be reversed with oxygen and ventilation, long-standing hypoxemia caused a persistent increase in PVR, thus the name of persistent pulmonary hypertension of the newborn (PPHN). Echocardiography is the best test to confirm PPHN. Following pre- and postductal saturation measurements that may indicate the presence of PPHN with the presence of severe hypoxemia, an echocardiography exam can be performed to confirm the diagnosis.

✓ Echocardiography is the best test to confirm PPHN.

✓ Inhaled nitric oxide is used to treat neonatal pulmonary hypertension.

Inhaled Nitric Oxide to Treat PPHN. Inhaled nitric oxide therapy is done to lower pulmonary vascular resistance. The walls of the pulmonary arteries are lined with endothelial cells that release a substance known as endothelium-derived relaxing factor (EDRF, which has been identified as inhaled nitric acid). This substance is responsible for vasodilation in vascular smooth muscle. EDRF has been found to be an essential link in the transition from fetal to neonatal circulation. Because iNO dilates the pulmonary arteries, it decreases the pulmonary vascular resistance and its associated hypertension. Once the pulmonary vessels are dilated, this change should improve the patient's pulmonary circulation, V/Q ratio, and oxygenation status.

CASE 15: HOME CARE AND DISEASE MANAGEMENT

INTRODUCTION

F.W. was a 66-year-old moderately obese white female weighing 109-Kg (240 lbs). She was brought to the emergency department by her husband and their 17-year-old daughter on a Saturday morning because they "couldn't keep her awake and her breathing sounded funny." Upon further questioning, the doctor was told by the patient's husband that she had no fever, but did have a productive cough with thick greenish sputum for the past 5 days. The patient had a history of COPD/asthma, cor pulmonale, hyponatremia, and hyperkalemia. She had no chest pain, except when coughing, but did have difficulty breathing when lying down. F.W.'s husband reported that she had no nausea and vomiting, and had not complained of pain other than when coughing.

Social history revealed that F.W. smoked one pack of cigarettes per day for 25 years, but quit 10 years ago. She did not drink alcohol. She lived with her spouse of 25 years and their 17-year-old daughter. Two other grown children from a previous marriage no longer lived at home.

✓ At the onset or worsening of dyspnea due to congestive heart failure or pulmonary congestion, breathing in an upright position (orthopnea) reduces pulmonary congestion and work of breathing.

Indication

Physical assessment revealed an obese white female, lethargic, with central cyanosis and in respiratory distress. From the information provided by her husband, the physician determined that the patient had experienced paroxysmal nocturnal dyspnea in addition to orthopnea. She was placed on a monitor that revealed these vital signs: blood pressure of 160/80 mm Hg, heart rate of 128/min, respiratory frequency of 28/min and labored, and SpO_2 of 85% on room air.

Paroxysmal nocturnal dyspnea and orthopnea are signs of congestive heart failure.

Further assessment showed 1+ to 2+ pedal edema, jugular vein distension, bibasilar crackles, and wheezes throughout.

Arterial blood gases on room air revealed:

Blood gases show acute ventilatory failure superimposed on chronic ventilatory failure. Noninvasive positive pressure ventilation is indicated for this patient.

pH	7.26
$PaCO_2$	88 mm Hg
PaO_2	38 mm Hg
HCO_3^-	38 mEq/L
SaO_2	80%

Pertinent lab work results were as follows:

WBC: 7.4 × 10^3 (*normal 3.2 to 9.8 ×* 10^3)
H&H: 16 Gm %/49.3% (*normal for women: hemoglobin 12 to 16 g/100 mL hematocrit 37 to 47%*)
Na^+: 119 (*normal 140 mEq/L*)

Initial Settings

IPAP provides mechanical ventilation and reduces the patient's work of breathing.

EPAP improves oxygenation and minimizes auto-PEEP due to air trapping.

The patient was stabilized in the emergency department prior to transfer to the telemetry unit. For her severe hypercapnia and hypoxemia, she was placed on nasal bilevel PAP at 12/6 cm H_2O with 5 L/min of oxygen. IPAP of 12 cm H_2O was used to augment the patient's ventilatory effort. EPAP of 6 cm H_2O and 5 L/min of oxygen were used to maintain oxygenation and minimize auto-PEEP due to air trapping.

Patient Management

IPAP of 12 cm H_2O is primarily responsible for the improvement of ventilation ($PaCO_2$ from 88 to 68 mm Hg).

For the excessive fluid buildup due to cor pulmonale, 60 mg of Lasix were given via an intravenous line. Blood was drawn for lab workup. A chest radiograph and repeat ABG on bilevel PAP were done. Lab reports showed normal WBC, hemoglobin, and hematocrit, and low sodium, which could explain some of her confusion. The chest radiograph showed enlarged heart (cardiomegaly) with pulmonary vascular congestion and bilateral infiltrates especially on the left side. (See Figure 19-7.)

Repeat ABG 2 hours after initiation of bilevel PAP showed:

EPAP of 6 cm H_2O and 5 L/min of oxygen is primarily responsible for the improvement of oxygenation (Pao_2 from 38 to 82 mm Hg).

pH	7.34
$PaCO_2$	68 mm Hg
PaO_2	82 mm Hg
HCO_3^-	36 mEq/L
SaO_2	96%
Bilevel PAP	12/6 cm H_2O
F_IO_2	5 L/min (nasal bilevel PAP)

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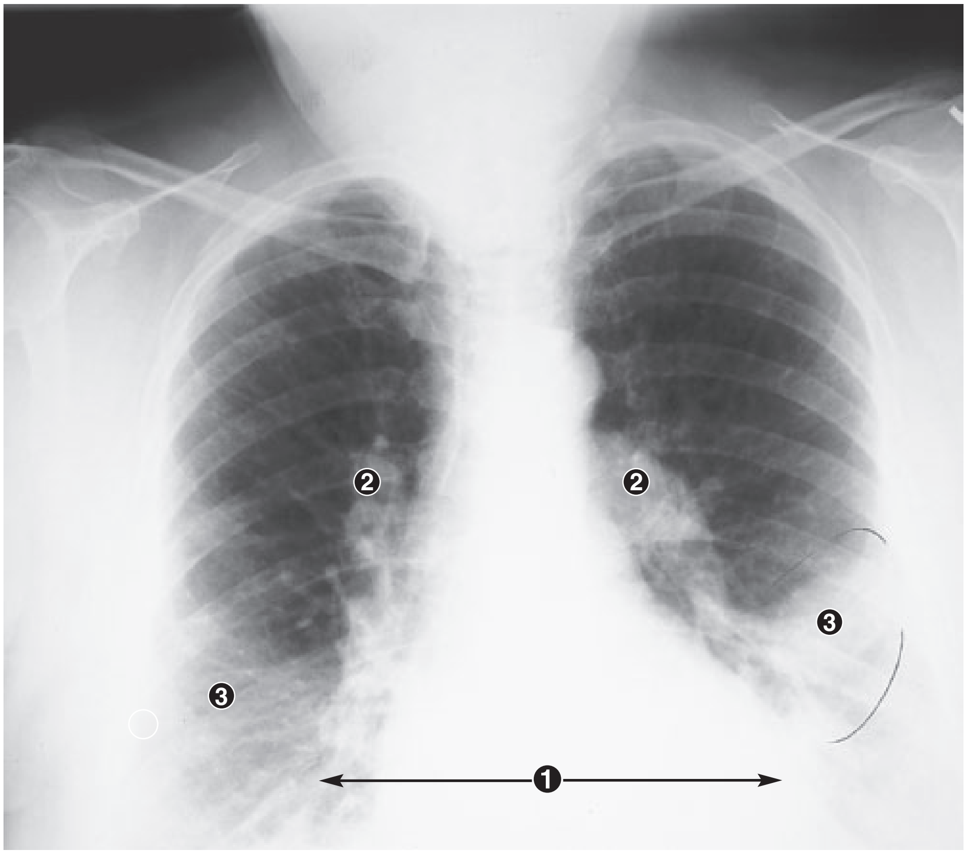

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FIGURE 19-7 Chest radiograph shows cardiomegaly (1); pulmonary vascular congestion (2); bilateral infiltrates (3).

Hospital Course

During the first 2 days of her hospital stay, F.W. was maintained on continuous nasal bilevel PAP at 12/6 cm H_2O with 2 L/min of oxygen. For the CHF and related conditions, she was treated with Lasix, Digoxin, and Cardizem. The pulmonologist ordered nebulizer treatments Q 4 hours with 2.5 mg albuterol and 0.5 mg ipratropium bromide in 3 mL of normal saline to relieve bronchospasm and promote secretion clearance. Prednisone (steroid) was ordered to decrease airway inflammation secondary to her asthma. Prophylactic antiobiotics (Biaxin) were administered, used to address her probable pneumonia since sputum induction was unsuccessful.

NOTE: F.W.'s medical record retrieved shortly after admission indicated that PFTs had been done 1 year before, and at that time her FEV_1 was 14% of predicted and her D_LCO was 21%. Baseline room air blood gases were:

pH	7.38
$PaCO_2$	67 mm Hg
PaO_2	45 mm Hg
HCO_3^-	38 mEq/L

On the third day following admission, the pulmonologist attempted to wean F.W. from bilevel PAP. She was placed on CPAP at 7.5 cm H_2O and ABGs were drawn an hour later; the results were as follows:

pH	7.16
$PaCO_2$	91 mm Hg
PaO_2	66 mm Hg
HCO_3^-	31 mEq/L
SaO_2	90%

CPAP alone increases the work of breathing. Patient requires some mechanical assistance via bilevel PAP (i.e., IPAP).

As a result of her deteriorating blood gas results and clinical condition, F.W. was placed back on bilevel PAP at 12/6 cm H_2O for 24 hours. Over the next several days, she was gradually weaned to 1 L/min via nasal cannula during the day and bilevel PAP at night. The physician discussed several options with F.W. and her family, and it was decided that upon discharge from the hospital she would use nocturnal ventilation at home. Her total hospital stay was 11 days.

Home Care Management

Twenty-four hours prior to discharge from the hospital, the case manager contacted a local home medical equipment provider for respiratory services and a home care agency for nursing services. Among other health care professionals, this case was reviewed with the respiratory therapist. Due to the complexity of her pulmonary condition and special management requirements, the physician decided to directly communicate with the respiratory therapist from the medical equipment company.

Arterial blood gases on day of discharge were as follows:

pH	7.32
$PaCO_2$	60 mm Hg
PaO_2	62 mm Hg
HCO_3^-	30 mEq/L
SaO_2	93%
Bilevel PAP	12/6 cm H_2O
Oxygen	1 L/min via nasal cannula during the day and bilevel PAP therapy at 12/6 cm H_2O, F_IO_2 of 24% at night

Orders for Home	Equipment
1. 1 L/min via nasal cannula continuously	Oxygen concentrator
2. Portable oxygen	Conserving device with M-6 cylinders
3. Nebulizer treatments QID	Small air compressor
4. Nocturnal bilevel PAP 10/5 cm H_2O, 24%	NPPV with backup frequency of 6/min
5. Keep head of bed elevated 30 degrees	Hospital bed
6. 1,200-calorie diet	
7. Ambulate as tolerated	
8. Incentive spirometry TID	Incentive spirometer (from hospital)

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Key Medications

1. 2.5 mg albuterol and 0.5 mg atrovent in 3.0 mL of 0.9% NaCl QID and prn for wheezing
2. Lasix 20 mg po BID
3. Prednisone po tapering dose
4. Cardizem po

Home Care Plan

The plan was devised by the physician and respiratory therapist. The patient was informed of the home care plan, as follows:

1. Maintain SpO_2 of 90% to 96%.
2. Maintain tidal volume of 650 mL to 900 mL while on NPPV.
3. Use backup frequency of 6/min to ensure adequate ventilation if patient were to become apneic.
4. Closely monitor patient for signs of CHF, hypercapnia, exacerbation of asthma/COPD, and pulmonary infection.
5. Notify physician of complications secondary to withdrawal or tapering of medications; specifically Prednisone and Lasix.
6. Increase exercise level as tolerated.
7. Improve diet and continue to lose weight. (F.W.'s height and weight: 5 ft 2 in and 235 lb)
8. Educate patient on disease and promote a healthier lifestyle (F.W. had already quit smoking 10 years ago).
9. Observe patient's attitude and family support and report to physician.

Patient Monitoring

F.W. required close monitoring for the first month. The same respiratory therapist visited her twice a week for the first 4 weeks and then once a week for the next 4 weeks. Routine monthly visits started in the third month.

NOTE: Nursing services were present in the home once a day for 2 weeks, and communication was established between the nurse (from a home care agency) and the respiratory therapist (from a DME company). During each visit, patient assessment was done and respiratory equipment was checked and serviced by the therapist.

Parameters monitored were as follows:

1. Tidal volume delivered via NPPV
2. Vital signs (BP, HR, f, SpO_2, weight, and breath sounds)

3. Bedside spirometry (PEFR, FVC, FEV_1) on initial visit, then every 6 months and prn
4. Pedal edema (present or not)
5. Exercise tolerance—using vital signs and subjective response as indicators
6. Saturation on room air—at rest and with exertion (walking with distance recorded)
7. Saturation on oxygen—at rest and with exertion (walking with distance recorded)
8. Sputum production—amount, color, and consistency
9. Subjective response from patient and family members
10. Patient compliance

NOTE: In order to have a successful outcome with home care, solid family support and proper emotional well-being of the patient are crucial. Motivation and a positive attitude on the part of the patient are essential in order to achieve a positive outcome. Any signs of patient noncompliance, lack of family support, depression, lack of necessary resources, or other emotional problems should be promptly reported to the physician so that these issues may be addressed. If a home care nurse is also seeing the patient, then all information should be shared so that the patient may receive the best quality care.

Patient Weekly Progress

Week 1. F.W. stayed in bed most of the time and got up only to go to the bathroom. Nocturnal bilevel ventilation was used at 10/5 cm H_2O with 1 L/min titrated through the nasal mask. Other parameters on the ventilator were: backup frequency 6/min, and expired tidal volume between 535 and 700 mL. Nebulizer treatments were done QID and incentive spirometry was done TID. Patient was able to achieve 800 to 1,000 mL on the incentive spirometry. Patient appeared to be compliant with her medications. Her weight was 235 lb with no signs of pedal edema. Vital signs were BP 150/84 mm Hg, f 24/min, HR 82/min, resting SpO_2 91% on 1 L/min and 88% on room air.

Patient had a positive attitude and excellent family support. She was excited about getting better and used the nocturnal ventilator 6 to 8 hours every night. She claimed that she was still very tired and sore from her hospital stay.

Week 2. F.W. had improved a great deal. She was walking to the kitchen and watching television in the living room. She remained on 10/5 cm H_2O of bilevel PAP with 1 L/min O_2 at night and 1 L/min per nasal cannula continuously during the day. Vital signs were: BP 130/68 mm Hg, f 16/min, HR 84/min, SpO_2 94% on 1 L/min resting and 89% to 90% on room air. Her weight was 218 lb. No pedal edema noted.

F.W. was in good spirits and compliant with regime.

Week 3. The expired tidal volume was 800 to 1,000 mL on 10/5 cm H_2O of bilevel PAP. This indicated an improvement of pulmonary mechanics. She also was able to

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reach 1,600 mL on her incentive spirometer TID. Her weight was down to 208 lb and she was ambulating more every week. No pedal edema noted.

Vital signs were BP 128/68 mm Hg, f 18/min, HR 70/min, SpO_2 97% on 1 L/min and 93% resting on room air. Ambulating SpO_2 was 88% on room air after walking just 25 ft, indicating that she continued to desaturate upon mild exertion.

Auscultation revealed bilateral expiratory wheezes. Small amounts of brownish sputum were noted. Bedside spirometry showed: PEFR 88 L/min, VC 1.69 L, FEV_1 0.709 L.

F.W. continued to improve and remained positive about improving the quality of her life.

Week 4. One month out of the hospital, F.W. remained stable. At this time she was able to go grocery shopping and have her hair done. She used her portable M-6 cylinders with a conserving device while she was out. According to both the patient and her spouse, her sleep had increased to 8 to 10 hours per night and she no longer took naps during the day. A decision was made to decrease her nocturnal ventilator pressures to 8/5 cm H_2O due to the increasing expired volumes (1,100 mL). On bilevel PAP of 8/5 cm H_2O, F.W. was more comfortable and had volumes of 650 to 850 mL.

NOTE: Tidal volumes were obtained with the patient in supine position and were monitored for 20 min. This allowed the patient to relax in order to obtain more accurate readings.

Vitals and physical signs: BP 100/50 mm Hg, f 20/min, HR 78/min, weight 211 lb, SpO_2 94% on 1 L/min. No pedal edema was noted. After ambulating 60 steps: HR 100, f28, SpO_2 86%. SpO_2 recovering within 3 min upon administration of 1 L/min O_2.

Complications

F.W. remained stable with no significant complications other than sinusitis for 6 months. During a routine visit almost 7 months after discharge, the therapist notified the physician with this assessment:

Vitals: BP 165/85 mm Hg, f 22/min, HR 80/min, weight 224 lb., SpO_2 91% on 1 L/min, 1 to 2+ pedal edema.
Auscultation: inspiratory and expiratory wheezes bilaterally (audible); diminished in bases bilaterally.
Tidal volume on bilevel PAP of 8/5 cm H_2O = 500 to 600 mL.

Patient's husband reported that she was not sleeping well at night and her legs were "jittering" during the night. He also stated that F.W. was falling asleep during her meals and she frequently complained of headaches.

The respiratory therapist increased the nocturnal ventilator pressures to 10/5 cm H_2O (expired volume of 800 to 1000 mL) and notified the physician promptly. An appointment was made to see the physician on the following day. As a result of the office visit, the Lasix dosage was increased. She no longer experienced the signs and symptoms that were present prior to the visit, thus avoiding a possible hospital admission.

Discussion

This case demonstrates one of the clinical indications for the use of nocturnal ventilation. F.W. was a COPD patient with hypercapnia and cor pulmonale. Is it also possible that F.W. suffered from a sleep disorder? Maybe; however, no sleep studies were ever done. F.W. qualified for nocturnal ventilation based on her $PaCO_2$ levels at the time of discharge from the hospital. Her pulmonary function studies indicated severe obstructive disease and poor diffusion capacity. Because of her obesity, F.W. also dealt with hypoventilation. The nocturnal ventilator assisted to overcome the chest wall compliance and pressure placed on the diaphragm by her large abdomen, thereby increasing volumes when F.W. was in supine position.

Without nocturnal ventilation, home oxygen, bronchodilator therapies via nebulizer, and oral medications, F.W. would probably have had numerous physician visits and even hospital stays. In fact, she has avoided ER visits and hospital admissions for over 1 year. The home therapy allowed her greater independence and helped her 'feel better;' therefore improving her quality of life. It is the responsibility of every home care provider to adequately assess patients, make sound clinical decisions, and report findings to the physician in a timely manner.

In this case the RCP quickly responded to signs and symptoms of hypercapnia and impending cardiac insufficiency. The pressure on the nocturnal ventilator was increased to improve minute ventilation and oxygenation, and the physician was consulted to increase Lasix to eliminate pulmonary vascular congestion due to heart failure.

CASE 16: END-OF-LIFE SEDATION ON MECHANICAL VENTILATION

INTRODUCTION

Mr. P.P. was a 66-year-old male with COPD and a smoking history of 80-pack years. About two years ago the patient noted a grayish patch on his tongue but did not immediately seek medical attention. He continued to smoke and use spitless tobacco. About five months ago the patient sought medical help after he developed the following symptoms: 1) a feeling that something was caught in the throat, 2) difficulty chewing or swallowing, 3) difficulty pronouncing words, and 4) numbness of the tongue.

Diagnosis and Initial Medications

Assessment and testing revealed oral squamous carcinoma of parts of the tongue and floor of the mouth which had metastasized to the cervical lymph nodes. He was treated with surgical resection of the tongue with extensive resection of bone and soft tissue. The malignancy progressed rapidly despite treatment and resulted in extensive tissue necrosis resulting in the following distressing symptoms: 1) strong nasal quality and loss of tongue made speech completely unintelligible, 2) extensive loss of teeth coupled with loss of tongue making it very difficult to swallow, 3) severe facial disfigurement, and 4) necrotic nonhealing oral ulcer causing severe malodor and facial pain.

Initially Mr. P's pain and symptoms were relatively well controlled with:

✓ Why are methadone, morphine sulfate, haloperidol, and lorazepam needed? The patient has multiple types of discomfort requiring different pharmacologic therapies.

- Methadone (50 mg thrice daily), a powerful synthetic opioid for long-term analgesia
- Immediate-release morphine sulfate (50 mg every 4 hours, prn) for severe pain
- Haloperidol (0.5 mg every 6 hours) for nausea and vomiting
- Lorazepam (0.5 mg every 4 hours) for anxiety

This regimen worked well for several weeks, but the pain worsened secondary to extensive local tissue necrosis from progression of the disease, leading to hospital admission for symptom control.

Pulse Oximetry and Pulse Co-Oximetry

SpO_2 89% on 3 L/min, SpHb 11.8 g/dL, and SpOC 14.5 mL O_2/dL (vol%) blood via noninvasive pulse oximeter.

Clinical Course

Numerous interventions were attempted to relieve Mr. Peace's pain, including:

✓ Changing to morphine sulfate administration by PCA allows the patient to participate in determining when dosing is needed and eliminates waiting for a nurse to deliver doses. While PCA imposes clinician-set limits on maximum dose per time it does not completely eliminate the possibility of overdose.

- Switching from methadone to continuous subcutaneous infusion of morphine (6 mg/h)
- Patient-controlled anesthesia (PCA) of morphine sulfate infusion 2 mg every 15 minutes as needed
- Lorazepam (0.5 mg every 4 hours)
- Metronidazole gel applied to the ulcerated tissue on the face (to control local infection and thereby the bad odor)
- Nasal cannula at 6 L/min; and a fan gently blowing on his face.

✓ It is important to monitor ventilation in spontaneously breathing patients when using high levels of sedation and/or analgesia. Capnography is a noninvasive means of monitoring ventilation.

Unfortunately, none of the treatments alleviated or attenuated his sense of severe pain. At this point, a family meeting was held to elicit goals of care and the following was determined:

- Mr. Peace adamantly refused further surgery, chemotherapy, and radiation therapy, and received complete support from his wife and adult children.
- Heroic life-prolonging measures (endotracheal intubation with mechanical ventilation, etc.) was discussed with Mr. Peace and his family; however, they

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elected to forgo ventilatory support and chose to continue with symptomatic therapy, invoking the Russell's do-not-resuscitate election in light of the futility of intubation and assisted ventilation.

- Patient and family refused a feeding tube.
- They elected for comfort care.

Heroic life-prolonging measures can mean more than intubation and mechanical ventilation. It is important to have the patient and health care surrogate decision maker define what heroic life-prolonging measures are desired.

Noninvasive ventilation could be a life-prolonging measure, but this patient's recent orofacial surgery makes even a nasal mask unlikely.

Over the next week, the patient's pain worsened despite aggressive pain management, and spontaneous ventilation is starting to worsen. The nasal cannula was changed to a high-flow nasal cannula (70% oxygen at 35 L/min BTPS), resulting in an SpO_2 of 93%, SpHb 11.7 g/dL, and SpOC 14.7 mL O_2/dL blood. The patient was able to rest more easily after implementation of the high-flow nasal cannula.

Midazolam, a benzodiazepine, was chosen to provide Mr. Peace's sedation because of its short half-life. There is extensive clinical experience with its use as sedative pharmacotherapy at the end of life.

Mr. Peace was clearly suffering greatly and this caused severe distress to his wife and children who could not bear to see him suffer in this manner. Since his pain was unendurable and refractory to all palliative measures, palliative sedation was proposed as a humane and compassionate approach to allay his suffering.

After explanation of the procedure, both he and his family readily agreed to deep and continuous palliative sedation. An informed consent document was signed, and a note describing the indications and plans for palliative sedation was recorded in the patient's medical record.

Phenobarbital is a cost-effective and efficacious agent that can be used as a first- or second-line medication and would have been added to Mr. Peace's regimen had a high dose (i.e., 120-200mg/d) of midazolam failed to provide adequate sedation.

End-of-Life Sedation

A 4-mg subcutaneous bolus of midazolam was then administered, followed by a continuous subcutaneous infusion of 1.5 mg of midazolam per hour. The Ramsay Sedation Scale was utilized to monitor depth of sedation, and the dosage of midazolam was titrated upward to maintain a deep level of sedation (a 4-mg bolus every 30–60 minutes, as needed, was utilized, with the continuous infusion increased by 0.5 mg/h after each bolus).

Propofol has also been touted as a valuable agent for palliative sedation; however, its cost and intravenous route of administration limit its use outside of an intensive care unit.

He was sedated within 10 minutes, but after 30 minutes he was still arousable with verbal stimulation and he complained of pain. A second bolus of midazolam was administered and his infusion increased to 2 mg/h.

Titration of midazolam continued over the next few hours until he was deeply sedated, with an eventual dose of 5 mg/h required to maintain deep and continuous sedation. He died 4 days later, sedated, peaceful, and with his family at his bedside.

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Appendix 1:

Respiratory Care Calculations

A. ARTERIAL OXYGEN TENSION TO INSPIRED OXYGEN CONCENTRATION (PaO_2/F_IO_2) INDEX

EQUATION

Arterial Oxygen Tension to Inspired Oxygen Concentration Index $= PaO_2/F_IO_2$

PaO_2/F_IO_2 Index: Arterial oxygen tension to inspired oxygen concentration index
PaO_2: Arterial oxygen tension in mm Hg
F_IO_2: Inspired Oxygen Concentration

NORMAL VALUE

(1) For weaning purpose with $F_IO_2 < 40\%$, 150 to 200 mm Hg or higher suggests a successful weaning outcome.

(2) For patients with non-cardiogenic pulmonary edema, the thresholds are <300 mm Hg for ALI and <200 mm Hg for ARDS.

EXAMPLE

Given: $PaO_2 = 50$ mm Hg, $F_IO_2 = 40\%$ (0.4)

$$PaO_2/F_IO_2 \text{ Index} = \frac{50 \text{ mm Hg}}{0.4}$$
$$= 125 \text{ mm Hg}$$

B. CARDIAC OUTPUT (CO): FICK'S ESTIMATED METHOD

EQUATION 1

$$CO = \frac{O_2 \text{ consumption}}{C_aO_2 - C_{\bar{v}}O_2}$$

EQUATION 2

$$CO = \frac{130 \times BSA}{C_aO_2 - C_{\bar{v}}O_2}$$

CO: Cardiac output in L/min (Q_T)
O_2 consumption: Estimated to be 130 × BSA, in mL/min $\dot{V}O_2$
C_aO_2: Arterial oxygen content in vol%
$C_{\bar{v}}O_2$: Mixed venous oxygen content in vol%
130: Estimated O_2 consumption rate of an adult, in mL/min/m^2
BSA: Body surface area in m^2

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NORMAL VALUE

CO = 4 to 8 L/min

EXAMPLE

Given: Body surface area	= 1.6 m^2
Arterial O_2 content	= 20 vol%
Mixed venous O_2 content	= 15 vol%

$$CO = \frac{O_2 \text{ consumption}}{C_aO_2 - C_{\bar{v}}O_2}$$

$$= \frac{130 \times BSA}{C_aO_2 - C_{\bar{v}}O_2}$$

$$= \frac{130 \times 1.6}{20\% - 15\%}$$

$$= \frac{208}{5\%}$$

$$= \frac{208}{0.05}$$

=4160 mL/min or 4.16 L/min

C. CEREBRAL PERFUSION PRESSURE

EQUATION

CPP = MAP − ICP

CPP: Cerebral Perfusion Pressure
MAP: Mean Arterial Pressure
ICP: Intracranial Pressure

NORMAL VALUE

70 to 80 mm Hg

EXAMPLE

Calculate the CPP given the following data: MAP = 90 mm Hg, ICP = 14 mm Hg. Is the calculated CPP within normal limits?

CPP = MAP − ICP
=90 mm Hg −14 mm Hg
=76 mm Hg

CPP is within normal limit of 70 to 80 mm Hg

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D. COMPLIANCE: DYNAMIC (C_{DYN})

EQUATION

$$C_{DYN} = \frac{\Delta V}{\Delta P}$$

C_{DYN}: Dynamic compliance in mL/cm H_2O
ΔV: Corrected tidal volume in mL
ΔP: Pressure change (Peak inspiratory pressure − PEEP) in cm H_2O

NORMAL VALUE

30 to 40 mL/cm H_2O

If the patient is intubated, use serial measurements to establish trend.

EXAMPLE

Given: $\Delta V = 500$ mL
Peak inspiratory pressure = 30 cm H_2O
PEEP = 10 cm H_2O

Calculate the dynamic compliance.

$$\begin{aligned} C_{DYN} &= \frac{\Delta V}{\Delta P} \\ &= \frac{500}{30 - 10} \\ &= \frac{500}{20} \\ &= 25 \text{ mL/cm } H_2O \end{aligned}$$

E. COMPLIANCE: STATIC (C_{ST})

EQUATION

$$C_{ST} = \frac{\Delta V}{\Delta P}$$

C_{ST}: Static compliance in mL/cm H_2O
ΔV: Corrected tidal volume in mL
ΔP: Pressure change (plateau pressure − PEEP) in cm H_2O

NORMAL VALUE

40 to 60 mL/cm H_2O

If the patient is intubated, use serial measurements to establish trend.

EXAMPLE

Given: $\Delta V = 500$ mL
Plateau pressure = 20 cm H_2O
PEEP = 5 cm H_2O

Calculate the static compliance.

$$C_{ST} = \frac{\Delta V}{\Delta P}$$

$$= \frac{500}{20 - 5}$$

$$= \frac{500}{15}$$

$$= 33.3 \text{ or } 33 \text{ mL/cm } H_2O$$

F. CORRECTED TIDAL VOLUME (V_T)

EQUATION

Corrected V_T = Expired V_T − Tubing Volume

Expired V_T: Expired tidal volume in mL
Tubing volume: Volume "lost" in tubing during inspiratory phase (Pressure change × tubing compression factor)

EXAMPLE

Expired V_T = 650 mL
Peak inspiratory pressure = 25 cm H_2O
Positive end-expiratory pressure (PEEP) = 5 cm H_2O
Tubing compression factor = 3 mL/cm H_2O

Calculate the corrected tidal volume.

$$\begin{aligned} \text{Tubing volume} &= \text{Pressure Change} \times 3 \text{ mL/cm } H_2O \\ &= (25 - 5) \text{ cm } H_2O \times 3 \text{ mL/cm } H_2O \\ &= 20 \times 3 \\ &= 60 \text{ mL} \end{aligned}$$

$$\begin{aligned} \text{Corrected } V_T &= \text{Expired } V_T - \text{Tubing Volume} \\ &= 650 - 60 \\ &= 590 \text{ mL} \end{aligned}$$

G. DEADSPACE TO TIDAL VOLUME RATIO (V_D/V_T)

EQUATION

$$\frac{V_D}{V_T} = \frac{(P_aCO_2 - P_{\bar{E}}CO_2)}{P_aCO_2}$$

V_D/V_T: Deadspace to tidal volume ratio in %
P_aCO_2: Arterial carbon dioxide tension in mm Hg
$P_{\bar{E}}CO_2$: Mixed expired carbon dioxide tension in mm Hg

NORMAL VALUE

20% to 40% in patients breathing spontaneously

40% to 60% in patients receiving mechanical ventilation

EXAMPLE

Given: P_aCO_2 = 40 mm Hg
$P_{\bar{E}}CO_2$ = 30 mm Hg

Calculate the V_D/V_T ratio.

$$
\begin{aligned}
\frac{V_D}{V_T} &= \frac{(P_aCO_2 - P_{\bar{E}}CO_2)}{P_aCO_2} \\
&= \frac{40 - 30}{40} \\
&= \frac{10}{40} \\
&= 0.25 \text{ or } 25\%
\end{aligned}
$$

H. I:E RATIO

EXAMPLE 1

When the *I* time and *E* time are known:

What is the *I:E* ratio if the inspiratory time is 0.4 sec and the expiratory time is 1.2 sec?

$$
\begin{aligned}
I{:}E &= \left(\frac{I\text{ time}}{I\text{ time}}\right) : \left(\frac{E\text{ time}}{I\text{ time}}\right) \\
&= \left(\frac{0.4}{0.4}\right) : \left(\frac{1.2}{0.4}\right) \\
&= 1 : 3
\end{aligned}
$$

EXAMPLE 2

When the *I* time % is known:

What is the *I:E* ratio if the inspiratory time ratio is 25% or 0.25?

$$
\begin{aligned}
I{:}E &= \left(\frac{I\text{ time}\%}{I\text{ time}\%}\right) : \left(\frac{1-I\text{ time}\%}{I\text{ time}\%}\right) \\
&= \left(\frac{0.25}{0.25}\right) : \left(\frac{1-0.25}{0.25}\right) \\
&= 1 : \left(\frac{0.75}{0.25}\right. \\
&= 1 :
\end{aligned}
$$

EXAMPLE 3

When the *I* time and f are known:

What is the *I:E* ratio if the inspiratory time is 1.5 sec and the frequency is 15/min?

I time $= 1.5$ sec

$$
\begin{aligned}
E\text{ time} &= \frac{60}{f} - I\text{ time} \\
&= \frac{60}{15} - 1.5 \\
&= 4 - 1.5 \\
&= 2.5\text{ sec} \\
I{:}E &= I\text{ time}: E\text{ tim} \\
&= 1.5 : 2.5
\end{aligned}
$$

$$= \left(\frac{1.5}{1.5}\right) : \left(\frac{2.5}{1.5}\right)$$
$$= 1 : 1.67$$

EXAMPLE 4

When the minute volume ($\dot{V}_E$) and flow rate are known:

Given: V_T = 800 mL (0.8 L)
f = 12/min
Flow rate = 40 L/min

What is the *I:E* ratio?

I:E ratio = (Minute volume) : (Flow rate – Minute volume)
$= (V_T \times f) : (\text{Flow rate} - V_T \times f)$
$= (0.8 \times 12) : (40 - 0.8 \times 12)$
$= 9.6 : (40 - 9.6)$
$= 9.6 : 30.4$
[divide both sides of this ratio by 9.6]
$= 1 : 3.2$

I. MEAN AIRWAY PRESSURE (mPaw)

EQUATION

$$\text{mPaw} = \left[\frac{\text{f} \times \text{I time}}{60}\right] \times (\text{PIP} - \text{PEEP}) + \text{PEEP}$$

mPaw: Mean airway pressure in cm H_2O
f: Respiratory frequency per minute
I time: Inspiratory time in sec
PIP: Peak inspiratory pressure in cm H_2O
PEEP: Positive end-expiratory pressure in cm H_2O

NORMAL VALUE

Below 30 cm H_2O (adults).

EXAMPLE

When PEEP is used.

Given: f = 45/min
I time = 0.5 sec
PIP = 35 cm H_2O
PEEP = 5 cm H_2O

Calculate the mean airway pressure.

$$\text{mPaw} = \left[\frac{\text{f} \times \text{I time}}{60}\right] \times (\text{PIP} - \text{PEEP}) + \text{PEEP}$$
$$= \left[\frac{45 \times 0.5}{60}\right] \times (35 - 5) + 5$$

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$$= \left[\frac{22.5}{60}\right] \times 30 + 5$$
$$= \{0.375\} \times 30 + 5$$
$$= 11.25 + 5$$
$$= 16.25 \text{ or } 16 \text{ cm } H_2O$$

J. MINUTE VENTILATION: EXPIRED AND ALVEOLAR

EQUATION 1

$$\dot{V}_E = V_T \times f$$

EQUATION 2

$$\dot{V}_A = (V_T - V_D) \times f$$

$\dot{V}_E$: Expired minute ventilation in L/min
$\dot{V}_A$: Alveolar minute ventilation in L/min
V_T: Tidal volume in mL
V_D: Deadspace volume in mL
f: Respiratory frequency per minute

EXAMPLE

Given: $V_T = 600$ mL
$V_D = 150$ mL
$f = 12$/min

Calculate the expired minute ventilation ($\dot{V}_E$) and the alveolar minute ventilation ($\dot{V}_A$).

$$\dot{V}_E = V_T \times f$$
$$= 600 \times 12$$
$$= 7200 \text{ mL/min or } 7.2 \text{ L/min}$$
$$\dot{V}_A = (V_T - V_D) \times f$$
$$= (600 - 150) \times 12$$
$$= 450 \times 12$$
$$= 5400 \text{ mL/min or } 5.4 \text{ L/min}$$

K. OXYGEN CONTENT: ARTERIAL (C_aO_2)

EQUATION

$$C_aO_2 = (Hb \times 1.34 \times S_aO_2) + (P_aO_2 \times 0.003)$$

C_aO_2: Arterial oxygen content in vol%
Hb: Hemoglobin content in g%
1.34: Amount of oxygen that fully saturated hemoglobin can hold in g
S_aO_2: Arterial oxygen saturation in %
P_aO_2: Arterial oxygen tension in mm Hg
0.003: Amount of dissolved oxygen for 1 mm Hg of P_aO_2

NORMAL VALUE

16 to 20 vol%

EXAMPLE

Given: Hb = 15 g%

$S_aO_2 = 98\%$

$P_aO_2 = 100$ mm Hg

Calculate the arterial oxygen content.

$$\begin{aligned}C_aO_2 &= (\text{Hb} \times 1.34 \times S_aO_2) + (P_aO_2 \times 0.003)\\ &= (15 \times 1.34 \times 98\%) + (100 \times 0.003)\\ &= 19.70 + 0.3\\ &= 20 \text{ vol\%}\end{aligned}$$

L. OXYGEN INDEX (OI)

EQUATION

$$\text{OI} = \frac{(\text{mPaw} \times F_IO_2)}{\text{PaO}_2}$$

OI: Oxygen index

mPaw: Mean airway pressure

F_IO_2: Inspired oxygen concentration in %

P_aO_2: Arterial oxygen tension in mm Hg

NORMAL VALUE

<30%

An oxygen index of 0.35 to 0.6 for 0.5 to 6 hours is inclusive for ECMO therapy.

EXAMPLE

Given : mPaw = 20 mm Hg, $F_IO_2 = 60\%$, $\text{PaO}_2 = 50$ mm Hg

$$\begin{aligned}\text{OI} &= \left(\frac{\text{mPaw} \times F_IO_2}{\text{PaO}_2}\right)\\ &= \left(\frac{20 \text{ mm Hg} \times 0.6}{50 \text{ mm Hg}}\right)\\ &= \frac{12 \text{ mm Hg}}{50 \text{ mm Hg}}\\ &= 0.24 \text{ or } 24\ \%\end{aligned}$$

M. SHUNT EQUATION (Q_{sp}/Q_T): CLASSIC PHYSIOLOGIC

EQUATION

$$\frac{Q_{SP}}{Q_T} = \frac{C_cO_2 - C_aO_2}{C_{c-}O_2 - C_{\bar{v}}O_2}$$

Q_{sp}/Q_T: Physiologic shunt to total perfusion ratio in %

C_cO_2: End-capillary oxygen content in vol%

C_aO_2: Arterial oxygen content in vol%

$C_{\bar{v}}O_2$: Mixed venous oxygen content in vol%

NORMAL VALUE

Less than 10%

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EXAMPLE

Given: $C_cO_2 = 20.4$ vol%
$C_aO_2 = 19.8$ vol%
$C_{\bar{v}}O_2 = 13.4$ vol%

$$Q_{sp}/Q_T = \frac{C_cO_2 - C_aO_2}{C_cO_2 - C_{\bar{v}}O_2}$$
$$= \frac{20.4 - 19.8}{20.4 - 13.4}$$
$$= \frac{0.6}{7}$$
$$= 0.086 \text{ or } 8.6\%$$

N. SHUNT EQUATION (Q_{sp}/Q_T): ESTIMATED

EQUATION 1

For non-critically ill individuals:

$$\frac{Q_{SP}}{Q_T} = \frac{C_cO_2 - C_aO_2}{5 + (C_cO_2 - C_aO_2)}$$

EQUATION 2

For critically ill (e.g., mechanical ventilation) patients:

$$\frac{Q_{SP}}{Q_T} = \frac{C_cO_2 - C_aO_2}{3.5 + (C_cO_2 - C_aO_2)}$$

Q_{sp}/Q_T: Physiologic shunt to total perfusion ratio in %
C_cO_2: End-capillary oxygen content in vol%
C_aO_2: Arterial oxygen content in vol%

NORMAL VALUE

Less than 10%

EXAMPLE 1

Given: Normal patient
$C_cO_2 = 20.4$ vol%
$C_aO_2 = 19.8$ vol%
Use 5 vol % as the estimated $C(a - \bar{v})O_2$and calculate Q_{sp}/Q_T.

$$\frac{Q_{SP}}{Q_T} = \frac{C_cO_2 - C_aO_2}{5 + (C_cO_2 - C_aO_2)}$$
$$= \frac{20.4 - 19.8}{5 + (20.4 - 19.8)}$$
$$= \frac{0.6}{5 + 0.6}$$
$$= \frac{0.6}{5.6}$$
$$= 0.107 \text{ or } 10.7\%$$

EXAMPLE 2

Given: A patient on mechanical ventilation

$C_cO_2 = 20.4$ vol %

$C_aO_2 = 19.8$ vol %

Use 3.5 vol % as the estimated $C(a-\overline{v})O_2$ and calculate $Q_{sp}\dot{Q}_T$.

$$\begin{aligned}\frac{Q_{SP}}{\dot{Q}_T} &= \frac{C_cO_2 - C_aO_2}{3.5 + (C_cO_2 \quad C_aO_2)}\\ &= \frac{20.4 - 19.8}{3.5 + (20.4-19.8)}\\ &= \frac{0.6}{3.5 + 0.6}\\ &= \frac{0.6}{4.1}\\ &= 0.146 \text{ or } 14.6\%\end{aligned}$$

O. VASCULAR RESISTANCE: PULMONARY

EQUATION

$$PVR = (\overline{PA} - PCWP) \times \frac{80}{CO}$$

PVR: Pulmonary vascular resistance in dyne • sec/cm^5

$\overline{PA}$: Mean pulmonary artery pressure in mm Hg

$PCWP$: Pulmonary capillary wedge pressure in mm Hg

80: Conversion factor from mm Hg/L/min to dynes • sec/cm^5

CO: Cardiac output in L/min($\dot{Q}_T$)

NORMAL VALUE

$PVR = 50$ to 150 dyne • sec/cm^5

EXAMPLE

A patient with pulmonary hypertension has the following measurements. What is the calculated pulmonary vascular resistance (PVR)?

$\overline{PA} = 22$ mm Hg

$PCWP = 6$ mm Hg

CO = 4.0 L/min

$$\begin{aligned}PVR &= (\overline{PA} - PCWP) \times \frac{80}{CO}\\ &= (22 - 6) \times \frac{80}{4.0}\\ &= 16 \times \frac{80}{4.0}\\ &= \frac{1280}{4}\\ &= 320 \text{ dyne} \cdot \text{sec/cm}^5\end{aligned}$$

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P. VASCULAR RESISTANCE: SYSTEMIC

EQUATION

$$SVR = (MAP - \overline{RA}) \times \frac{80}{CO}$$

SVR: Systemic vascular resistance in dyne • sec/cm^5
MAP: Mean arterial pressure in mm Hg*
$\overline{RA}$: Mean right atrial pressure in mm Hg
80: Conversion factor from mm Hg/L/min to dynes • sec/cm^5
CO: Cardiac output in L/min ($\dot{Q}_T$)

NORMAL VALUE

SVR = 800 to 1,500 dyne • sec/cm^5

EXAMPLE

A patient has the following measurements. What is the calculated systemic vascular resistance (SVR)?

MAP = 70 mm Hg
$\overline{RA}$ = 8 mm Hg
CO = 5.0 L/min

$$\begin{aligned} SVR &= (MAP - \overline{RA}) \times \frac{80}{CO} \\ &= (70 - 8) \times \frac{80}{5.0} \\ &= 62 \times \frac{80}{5.0} \\ &= \frac{4{,}960}{5} \\ &= 992 \text{ dyne·sec/cm}^5 \end{aligned}$$

Q. VENTILATOR RATE NEEDED FOR A DESIRED PaCO$_2$

EQUATION 1

$$\text{New Rate} = \frac{\text{Rate} \times P_aCO_2}{\text{Desired } P_aCO_2}$$

EQUATION 2

$$\text{New Rate} = \frac{(\text{Rate} \times P_aCO_2) \times (V_T - V_D)}{\text{Desired } P_aCO_2 \times (\text{New } V_T - \text{New } V_D)}$$

New rate: Ventilator rate needed for a desired PaCO$_2$
Rate: Original ventilator rate per minute
PaCO$_2$: Original arterial carbon dioxide tension in mm Hg
Desired PaCO$_2$: Desired arterial carbon dioxide tension in mm Hg
V_T: Original tidal volume
V_D: Original deadspace volume

New V_T: New tidal volume
New V_D: New deadspace volume

NORMAL VALUE

Set rate to provide eucapnic (patient's normal) ventilation.

EXAMPLE

The $PaCO_2$ of a patient is 55 mm Hg at a ventilator rate of 10/min. What should be the ventilator rate if a $PaCO_2$ of 40 mm Hg is desired, assuming the ventilator tidal volume and spontaneous ventilation are stable?

$$\begin{aligned}\text{New Rate} &= \frac{(\text{Rate} \times P_aCO_2)}{\text{Desired } P_aCO_2} \\ &= \frac{(10 \times 55)}{40} \\ &= \frac{550}{40} \\ &= 13.75 \text{ or } 14/\text{min}\end{aligned}$$

R. WEANING INDEX: RAPID SHALLOW BREATHING

EQUATION

$$\text{Rapid Shallow Breathing Index} = \frac{f}{V_T}$$

f: Spontaneous respiratory frequency/min (without or with minimal pressure support)
V_T: Spontaneous tidal volume (in liters)

NORMAL VALUE

$<$100 breaths/min/L is predictive of weaning success

EXAMPLE

Calculate the rapid shallow breathing index, given that the spontaneous respiratory frequency and tidal volume are 14 breaths/min and 0.5 L (500 mL), respectively. Does this index indicate a successful weaning outcome?

$$\begin{aligned}\text{Rapid Shallow Breathing Index} &= \frac{f}{V_T} \\ &= \frac{14}{0.5} \\ &= 28 \text{ breaths/min/L}\end{aligned}$$

Since the rapid breathing index is less than 100, it indicates a successful weaning outcome.

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Appendix 2:

Normal Electrolyte Concentrations in Plasma

Normal Electrolyte Concentrations in Plasma

	Concentration (Range) mEq/L	**Anions**	**Concentration Cations (Range) mEq/L**
Na^+	140 (138 to 142)	Cl^-	103 (101 to 105)
K^+	4 (3 to 5)	HCO_3^-	25 (23 to 27)
Ca^{++}	5 (4.5 to 5.5)	Protein	16 (14 to 18)
Mg^{++}	2 (1.5 to 2.5)	HPO_4^{--}, $H_2PO_4^-$	2 (1.5 to 2.5)
		SO_4^{--}	1 (0.8 to 1.2)
		Organic acids	4 (3.5 to 4.5)
Total	151	Total	151

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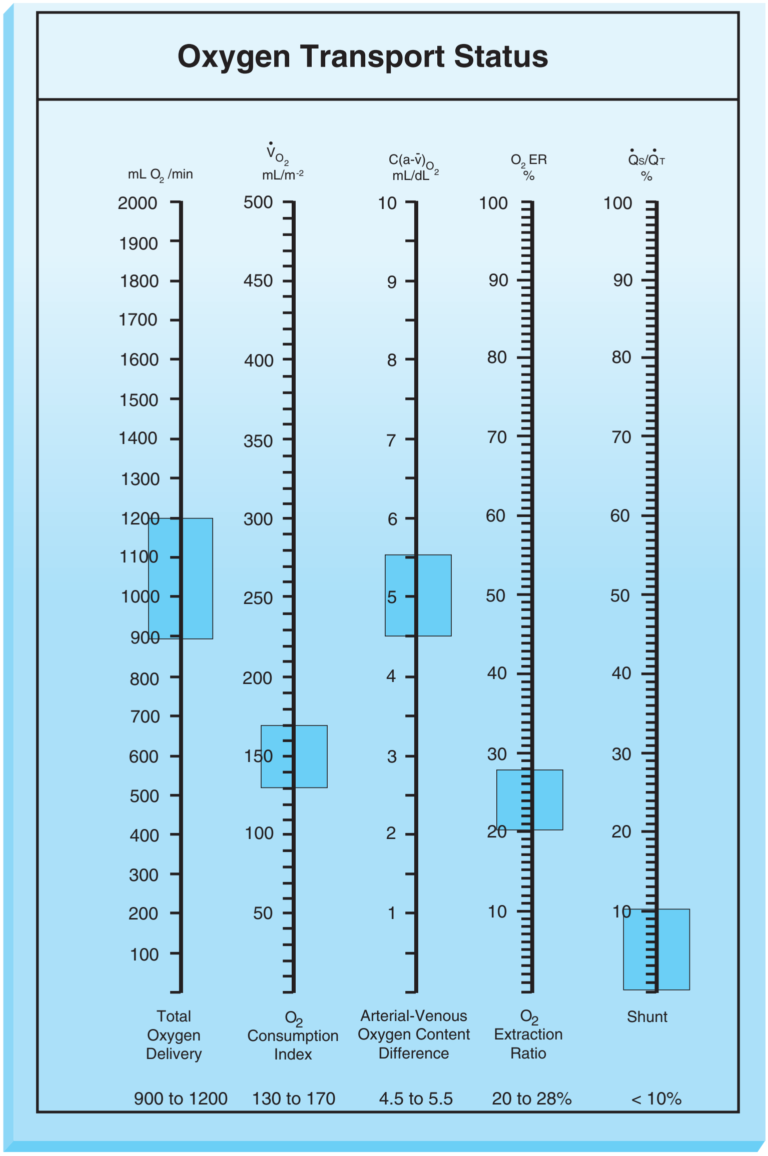
Appendix 3:

Oxygen Transport Normal Ranges



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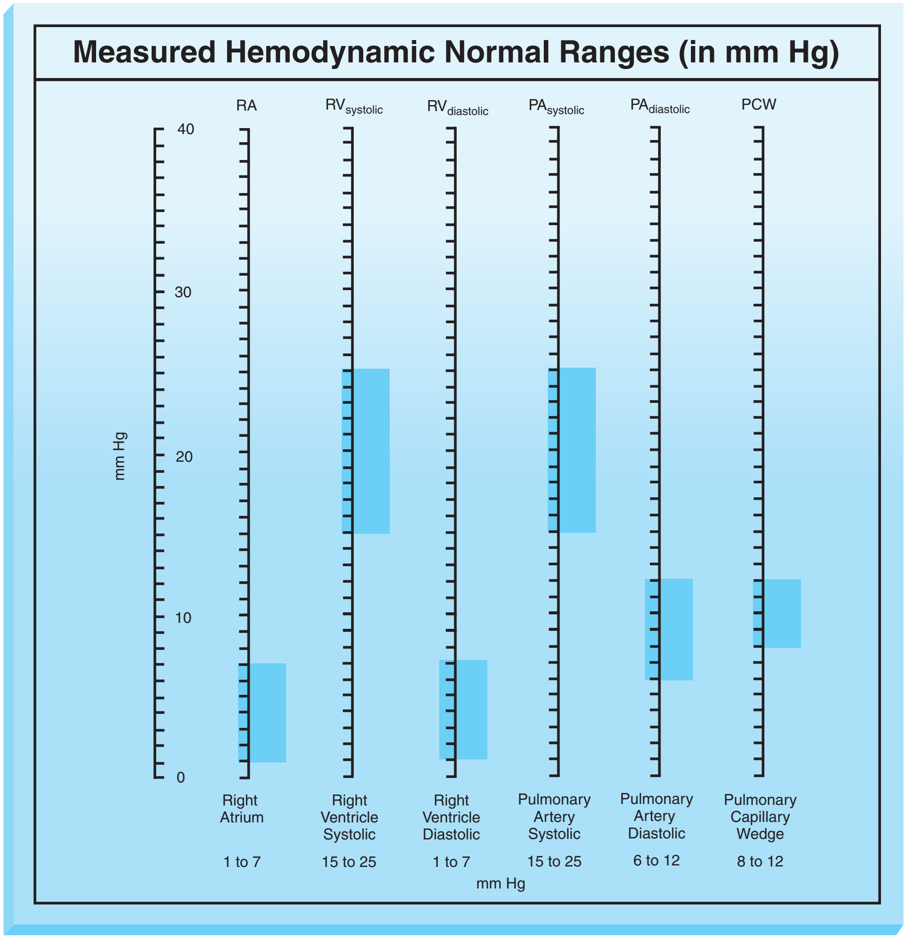
Appendix 4:

Hemodynamic Normal Ranges

Hemodynamic Values Directly Obtained by Means of the Pulmonary Artery Catheter

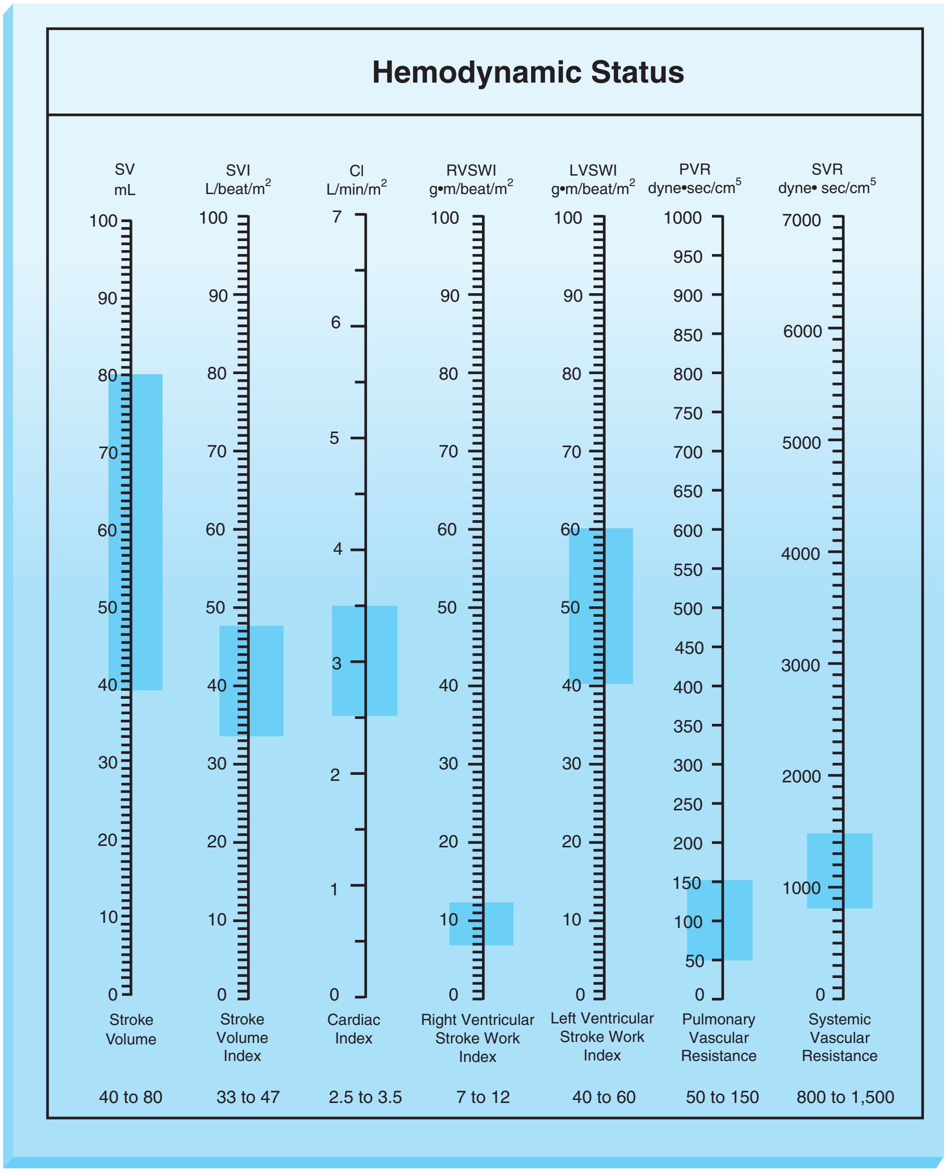
Hemodynamic Value	Abbreviation	Normal Range
Central venous pressure	CVP	1 to 7 mm Hg
Right atrial pressure	RAP	1 to 7 mm Hg
Mean pulmonary artery pressure	$\overline{\text{PAP}}$	15 mm Hg
Pulmonary capillary wedge pressure	PCWP	8 to 12 mm Hg
Cardiac output	CO	4 to 8 L/min



Adapted from Des Jardins, T.R. *Cardiopulmonary Anatomy and Physiology: Essentials for Respiratory Care*, 5th ed. Clifton Park, NY: Delmar Cengage Learning, 2008.

Computed Hemodynamic Values

Hemodynamic Variable	Abbreviation	Normal Range
Stroke volume	SV	40 to 80 mL
Stroke volume index	SVI	33 to 47 L/beat/m^2
Cardiac index	CI	2.5 to 3.5 L/min/m^2
Right ventricular stroke work index	RVSWI	7 to 12 g·m/beat/m^2
Left ventricular stroke work index	LVSWI	40 to 60 g·m/beat/m^2
Pulmonary vascular resistance	PVR	50 to 150 dyne·sec/cm^5
Systemic vascular resistance	SVR	800 to 1500 dyne·sec/cm^5



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Appendix 5:

Glasgow Coma Score

Glasgow coma score (GCS) ranges from 3 to 15 and is composed of three parameters: best eye response, best verbal response, and best motor response. The GCS is typically reported in its components such as E2 V1 M3 = GCS 6. A GCS score of 13 to 14 correlates with a mild brain inju-ry, 9 to 12 suggests a moderate injury, and 8 or less a severe brain injury.

BEST EYE RESPONSE (1 TO 4)

1. No eye opening
2. Eye opening to pain
3. Eye opening to verbal command
4. Eyes open spontaneously

BEST VERBAL RESPONSE (1 TO 5)

1. No verbal response
2. Incomprehensible sounds
3. Inappropriate words
4. Confused
5. Orientated

BEST MOTOR RESPONSE (1 TO 6)

1. No motor response
2. Extension to pain
3. Flexion to pain
4. Withdrawal from pain
5. Localizing pain
6. Obeys commands

REFERENCE

Teasdale, G. and Jennett, B. (1974). "Assessment of coma and impaired consciousness: A practical scale," *The Lancet, 304*, 81–84.

Appendix 6:

Apache II Severity of Disease Classification System*

	High Abnormal Range						Low Abnormal Range			
Physiologic Variable	**+4**	**+3**	**+2**	**+1**	**0**	**+1**	**+2**	**+3**	**+4**	**Points**
Temperature—rectal (°C)	≥41°	39 to 40.9°		38.5 to 38.9°	36 to 38.4°	34 to 35.9°	32 to 33.9°	30 to 31.9°	≤29.9°	
Mean arterial pressure—mm Hg	≥160	130 to 159	110 to 129		70 to 109		50 to 69		≤49	
Heart rate (ventricular response)	≥180	140 to 179	110 to 139		70 to 109		55 to 69	40 to 54	≤39	
Respiratory rate (non-ventilated or ventilated)	≥50	35 to 49		25 to 34	12 to 24	10 to 11	6 to 9		≤5	
Oxygenation: A-aDO$_2$ or PaO$_2$ (mm Hg) a. F$_I$O$_2$ ≥0.5 record A-aDO$_2$	≥500	350 to 499	200 to 349		<200					
b. F$_I$O$_2$ <0.5 record PaO$_2$					PO$_2$>70	PO$_2$ 61 to 70		PO$_2$ 55 to 60	PO$_2$ <55	
Arterial pH **(preferred)**	≥7.7	7.6 to 7.69		7.5 to 7.59	7.33 to 7.49		7.25 to 7.32	7.15 to 7.24	<7.15	
Serum HCO$_3^-$ (venous mEq/l) **(not preferred, but may use if no ABGs)**	≥52	41 to 51.9		32 to 40.9	22 to 31.9		18 to 21.9	15 to 17.9	<15	
Serum sodium (mEq/l)	≥180	160 to 179	155 to 159	150 to 154	130 to 149		120 to 129	111 to 119	≤110	
Serum potassium (mEq/l)	≥7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5	
Serum creatinine (mg/dl) Double point score for acute renal failure	≥3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4		<0.6			
Hematocrit (%)	≥60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20	
White blood count (total/mm^3) (in 1000s)	≥40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1	
Glasgow coma score (GCS) Score = 15 minus actual GCS										

(*continues*)

A. Total Acute Physiology Score (sum of 12 above points)					
B. Age points (years)	≤44 = 0;	45 to 54 = 2;	55 to 64 = 3;	65 to 74 = 5;	≥75 = 6
C. Chronic Health Points (see below)					
Total APACHE II Score (add together the points from A+B+C)					

Chronic Health Points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

5 points for nonoperative or emergency postoperative patients

2 points for elective postoperative patients

Definitions: Organ insufficiency or immunocompromised state must have been evident **prior** to this hospital admission and conform to the following criteria: **Liver**—biopsy proven cirrhosis and documented portal hypertension; episodes of past upper GI bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma. **Cardiovascular**—New York Heart Association Class IV. **Respiratory**—Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mm Hg), or ventilator dependency. **Renal**—receiving chronic dialysis. **Immunocompromised**—the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

Interpretation of Score:			
0 to 4 = ~4% death rate	10 to 14 = ~15% death rate	20 to 24 = ~40% death rate	30 to 34 = ~75% death rate
5 to 9 = ~8% death rate	15 to 19 = ~25% death rate	25 to 29 = ~55% death rate	Over 34 = ~85% death rate

*Adapted from *Crit Care Med* 1985; 13:818–829.

Glossary

acetylcholine: An ester that plays a role in the transmission of nerve impulses at synapses and neuromuscular junctions. It is metabolized by an enzyme, cholinesterase. Too much or too little acetylcholine at the motor endplates may lead to muscle blockade.

acute lung injury: A condition of sudden onset, characterized by non-cardiogenic pulmonary edema on chest radiograph and a PaO_2/F_IO_2 of $\leq$ 300 mm Hg.

acute respiratory distress syndrome: A condition of sudden onset, characterized by non-cardiogenic pulmonary edema on chest radiograph and a PaO_2/F_IO_2 of $\leq$ 200 mm Hg.

acute ventilatory failure: An increase of $PaCO_2$ ($>$50 mm Hg) with a concurrent decrease of arterial (pH $<$7.30).

adaptive pressure control: A mode of pressure-controlled breath that utilizes closed-loop control of the pressure setting to maintain a minimum delivered tidal volume.

adaptive support ventilation (ASV): A mode of ventilation that changes the number of mandatory breaths and pressure support level according to the patient's breathing pattern.

afterload: The resistance of the blood vessels into which the ventricle is pumping blood.

airway resistance: The degree of airflow obstruction in the airways.

airway opening pressure (P_{AO}): Sum of transairway pressure (P_{TA}) and alveolar pressure (P_{ALV}).

airway pressure release ventilation (APRV): A mode of ventilation in which the spontaneous breaths are at an elevated baseline (i.e., CPAP). This elevated baseline is periodically "released" to facilitate expiration.

alarm: An absolute value of a parameter on the ventilator beyond which an alert is invoked to warn that the safety limit has been breached.

alveolar deadspace: The normal lung volume that has become unable to take part in gas exchange because of reduction or lack of pulmonary perfusion (e.g., pulmonary embolism).

alveolar pressure (P_{ALV}): Pressure required to overcome the elastic recoil property of the lungs.

alveolar volume: The portion of tidal volume that takes part in gas exchange.

alveolar-arterial oxygen pressure gradient [$P_{(A-a)}O_2$]: The difference of P_AO_2 and PaO_2. A gradient over 450 mm Hg while on 100% oxygen indicates severe hypoxemia or intrapulmonary shunting.

anatomic deadspace: The volume occupying the conducting airways that does not take part in gas exchange (estimated to be 1 mL/lb ideal body weight).

anion gap: The difference between cations (positive ions) and anions (negative ions) in the plasma. The normal range is 15–20 mEq/L when K^+ is included in the calculation (10 to 14 mEq/L when K^+ is excluded).

antiemetic: Preventing nausea and vomiting.

anxiolysis: Diminishing anxiety.

apnea index: Average number of apneas in each hour of sleep during a test.

apnea-hypopnea index: Average number of apnea and hypopnea in each hour of sleep during a test.

assist/control (AC): In the assist/control (AC) mode, the patient may increase the frequency (assist) in addition to the preset mechanical frequency (control). Each assist breath provides the preset mechanical tidal volume.

atropine sulfate: A medication used to reduce vagal response, oral secretions, and bronchospasm during bronchoscopy.

autoclave: A method of sterilization using steam pressure, usually at 250°F (121°C) for a specific length of time. (Up to 275°F or 135°C for reusable LMAs.)

automatic tube compensation (ATC): A mode of ventilation that offsets and compensates for the airflow resistance imposed by the artificial airway.

automode: This mode provides time-triggered, PRVC breaths when prolonged apnea is detected (12, 8, and 5 sec in adult, pediatric, and neonatal modes, respectively).

auto-PEEP: Unintentional PEEP associated with pressure support ventilation, high tidal volume and frequency, inadequate inspiratory flow, excessive I-time, inadequate E-time, and air trapping.

barbiturates: A group of drugs that depress the central nervous system. Adverse effects are many, including alteration of the respiration, heart rate, blood pressure, and temperature. They are used in seizure disorders, control of elevated intracranial pressure.

barotrauma (volutrauma): Air leak into the pleural space caused by excessive pressure or volume in the lung parenchyma.

benzodiazepines: A group of drugs with strong hypnotic and sedative actions; used mainly to reduce anxiety and to induce sleep.

bilevel positive airway pressure (BiPAP): An airway pressure strategy that applies independent positive airway pressures (PAP) to both inspiration and expiration.

biphasic positive airway pressure (BiPAP): A mode that has two baseline pressures (P_{INSP} and PEEP). It allows spontaneous breathing at any point in the mechanical ventilation cycle.

blind distal end: The far end of a tube without an opening.

blind intubation: Insertion of an artificial airway without use of visual aid or under direct vision.

brachial plexopathy: Decreased movement or sensation in the arm and shoulder.

bronchial brushing: Tissue or loosened cell specimens collected by a shielded small brush using a brushing motion.

carbon dioxide elimination ($\dot{V}CO_2$): A technology to monitor and measure cardiac output based on changes in respiratory CO_2 concentration during a period of rebreathing.

cardiac index (CI): A cardiac output measurement relative to a person's body size.

cardiac output: Blood volume pumped by the heart in 1 min. Normal range is 4–8 L/min.

carina: The point at the lower end of the trachea separating openings of the main-stem bronchi.

cathartic agents: Active purgatives used to produce bowel movements.

central venous pressure (CVP): Pressure measured in the vena cava or right atrium. It reflects the status of blood volume in the systemic circulation. Right ventricular preload.

cerebral perfusion pressure (CPP): Pressure required to provide blood flow, oxygen, and metabolite to the brain. It is a function of mean arterial pressure (MAP) and intracranial pressure (ICP). $CPP = MAP - ICP$. Normal range = 70–80 mm Hg.

chest tube (thoracostomy tube): A tube that connects the pleural space and drainage system for removal of air or fluid.

chronotropic: Affecting the heart rate.

circuit compressible volume: Expansion of the ventilator circuits during inspiration leading to a small "lost" volume of gas that does not reach the patient, but is recorded as part of the expired tidal volume.

clinical pulmonary infection score: An objective scoring system to use as an additional aid in the diagnosis of ventilator-associated pneumonia (VAP) and decision on antimicrobial therapy.

compression factor: The amount of expansion of the ventilator circuit or humidifier during the inspiratory phase measured in mL/cm H_2O. This volume is considered "lost" and unavailable to the patient.

compressor: A device capable of building up pressure by compressing the volume of air.

constant flow waveform (CFW): Flow-time waveform where the peak flow occurs at or near beginning inspiration and remains constant until end-inspiration.

continuous positive airway pressure (CPAP): CPAP is PEEP applied to the airway of a patient who is breathing spontaneously. It is used to treat refractory hypoxemia in patients who are able to maintain adequate spontaneous ventilation.

contractility: Pumping strength of the heart. Contractility may be increased by improving the blood volume or by positive inotropic medications.

control mode: In control mode, the ventilator delivers the preset tidal volume at a set time interval (time-triggered frequency).

controlled mandatory ventilation (CMV): Time-triggered mandatory breaths provided by the ventilator. Also called controlled mechanical ventilation or continuous mandatory ventilation.

controller: The mechanism that provides a mode of ventilation within a specific parameter (pressure, time, volume, or flow).

corticosteroids: Hormones that are released from the cortex of the adrenal gland. Their potent anti-inflammatory effects make corticosteroids useful in the treatment of asthma and chronic bronchitis. Corticosteroids are available for intravenous administration as well as inhalation. Examples of inhaled steroids are dexamethasone (Decadron, Respihaler), beclomethasone (Beclovent, Vanceril), flunisolide (AeroBid), and triamcinolone (Azmacort).

culture and sensitivity: A laboratory procedure that grows the microbes in a medium and tests their sensitivity or resistance to different antimicrobial drugs.

cycle variable: A measurement that causes the breath to end.

deadspace ventilation: Ventilation in excess of perfusion; wasted ventilation.

decremental recruitment maneuver: A method of titration for optimal PEEP by setting a high CPAP (and PEEP) and gradually decreasing the pressure and F_IO_2.

depolarizing agents: Drugs that prolong the depolarization phase of muscle contraction, thus rendering the repolarization/depolarization sequence (normal mechanism for muscle movement) impossible and causing muscle blockade. An example is succinylcholine (Anectine, Quelicin).

desaturation index: Average number of oxygen desaturations of 4% or more from baseline in each hour of sleep during a test.

descending ramp flow waveform (DRFW): Flow-time waveform where the peak flow occurs at or near beginning inspiration and decreases to baseline at end-inspiration.

dexmedetomidine (Precedex): An intravenous drug that offers anxiolysis and analgesia but no respiratory depression.

diffusion defect: Pathologic condition leading to impaired gas exchange through the alveolar-capillary membrane (e.g., interstitial or pulmonary edema).

double-lumen endobronchial tube: A special airway for independent lung ventilation. It has two separate lumens, two cuffs, and two pilot balloons.

dual control mode: A combined mode between two control variables (e.g., pressure and volume) that are regulated by independent feedback loops so that the delivered breath switches between pressure-controlled and volume-controlled.

dyshemoglobins: Hemoglobins that do not carry oxygen (e.g., carboxyhemoglobin, methemoglobin). In the presence of dyshemoglobins, the pulse oximeter reads higher than actual SaO_2.

endotracheal tubes: An artificial airway inside the trachea that is inserted through the mouth or nostril.

end-tidal carbon dioxide monitoring: The CO_2 level measured at the end of exhalation; measured in mm Hg.

esophageal gastric tube airway (EGTA): A tube used in esophageal intubation. It has a patent distal end to relieve gastric distention.

esophageal obturator airway (EOA): A tube that has a closed (blind) distal end and it is inserted into the esophagus.

esophageal-tracheal combitube (ETC): An airway that may be inserted into the esophagus or trachea.

eucapnic ventilation: The amount of ventilation to provide and maintain a patient's normal $PaCO_2$.

expiratory positive airway pressure: An airway pressure that is above 0 cm H_2O at end-expiration.

expiratory positive airway pressure (EPAP): An airway pressure that is above 0 cm H_2O during the expiratory phase of a respiratory cycle.

expiratory time (T_E): Time period from beginning expiration to beginning inspiration of next mechanical or spontaneous breath.

extracellular fluid (ECF): Fluid in the plasma and interstitial space. It accounts for 20% of total body water and is mainly affected by the sodium concentration in the plasma.

extracorporeal membrane oxygenation (ECMO): Oxygenation of blood outside the body through a membrane oxygenator.

fiberoptic bronchoscope: An instrument that uses glass fibers to transmit images of the airway for diagnostic or therapeutic procedures under direct vision.

flow rate: Peak flow during the inspiratory phase. It determines how fast the tidal volume is delivered to the patient.

flow-triggered: Flow-triggering strategy uses a combination of continuous flow and demand flow. Before inspiration, the delivered flow equals the return flow. As the patient initiates a breath, the return flow to the ventilator is decreased, and this flow differential triggers a mechanical breath.

flow-volume loop (FVL): Graphic display of changes in flow and volume during a complete respiratory cycle.

forceps biopsy: Tissue specimens collected by the forceps located at the distal end of the insertion tube.

full-face mask: An interface that covers almost the entire face of a patient.

gamma-aminobutyric acid (GABA): A major central nervous system inhibitory transmitter that regulates the chloride ion channel and hyperpolarizes the neurons. Once the neurons are hyperpolarized and become resistant to repeated depolarization, sedation results.

gastrointestinal (GI): Organ system including the stomach and intestines.

Glasgow coma scale: A scoring system to determine the degree of traumatic brain injury; score of 13 to 14 (mild), score of 9 to 12 (moderate), score of 3 to 8 (severe).

Gram stain: A method for staining bacteria. Gram-positive bacteria (e.g., *Staphlococcus*) retain the gentian violet (purple) color, and gram-negative bacteria (e.g., *Pseudomonas*) take the red counterstain.

haloperidol (Haldol): A drug for the control of delirium in mechanically ventilated patients.

hemodynamic monitoring: Measurement of the blood pressure in the vessels or heart chambers during contraction (systole) and relaxation (diastole).

hepatic perfusion: Blood flow to the liver. It is decreased when the blood volume or cardiac output is low.

high frequency jet ventilation (HFJV): Ventilation through a specially designed endotracheal tube, generally at a frequency between 240 and 660/min (4 to 11 Hz).

high frequency oscillatory ventilation (HFOV): Ventilation produced by a piston pump or loudspeaker, usually at a frequency between 480 and 1,800/min.

high frequency ventilation (HFV): A type of ventilation that uses very high frequencies. It is subdivided into three categories: high frequency positive pressure ventilation (60 to 150 cycles per minute); high frequency jet ventilation (240 to 660 cycles per minute); and high frequency oscillatory ventilation (480 to 1,800 cycles per minute).

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hyperbaric condition: An environment in which the atmospheric pressure is greater than one barometric pressure (e.g., diving under water, hyperbaric chamber).

hyperbaric oxygen: Supplemental oxygen under hyperbaric conditions; used in conditions such as severe carbon monoxide poisoning, decompression sickness, gas gangrene, and anaerobic infections.

hypobaric condition: An environment in which the atmospheric pressure is less than one barometric pressure (e.g., high altitude, airplane cabin in flight).

hypopnea: Reduction in airflow for 10 or more seconds that is at least 50% below an estimated baseline amplitude, usually associated with oxygen desaturation or pulse alteration.

hypoventilation: Below normal level of alveolar ventilation characterized by an elevated $PaCO_2$.

hypoxemia: Deficiency of oxygen in blood; low PaO_2.

hypoxia: Deficiency of oxygen in tissues.

hypoxic hypoxia: Lack of oxygen in the organs and tissues due to a reduction in inspired oxygen tension.

hypoxic-ischemic encephalopathy (HIE): A condition caused by a severe lack of oxygen supply to the brain leading to damage to the cells and neurons of the brain and spinal cord.

I:E ratio: A time ratio comparing the inspiratory time and expiratory time, normally between 1:2 and 1:4 in mechanical ventilation. This ratio is regulated by the inspiratory flow rate, I time, or E time and is affected by the tidal volume and respiratory rate.

impedance cardiography (ICG): A noninvasive procedure to measure or trend the hemodynamic status of a patient.

impending ventilatory failure: A gradual increase of $PaCO_2$ (>50 mm Hg) caused by deteriorating lung functions.

inotropic: Affecting the contraction.

inspiratory positive airway pressure (IPAP): An airway pressure that is above 0 cm H_2O during the inspiratory phase of a respiratory cycle.

inspiratory time: Time from beginning inspiration to end of inspiration/beginning expiration.

interhospital transport: Moving a patient between two hospitals.

intermittent mandatory ventilation (IMV): A mode in which the ventilator delivers control (mandatory) breaths and allows the patient to breathe spontaneously to any tidal volume the patient is capable of between the mandatory breaths.

intra-abdominal pressure (IAP): Pressure measured by a transducer via a transurethral bladder catheter.

intracellular fluid (ICF): Fluid within the cells. It accounts for 40% of total body water.

intrahospital transport: Moving a patient between two locations within the hospital.

intrapulmonary shunting: Pulmonary blood flow in excess of ventilation; wasted perfusion (e.g., atelectasis).

inverse ratio pressure-controlled ventilation (IRPCV): A mode of ventilation in which the peak inspiratory pressure is preset and the I time is longer than the E time. Same as pressure-controlled inverse ratio ventilation (PC-IRV).

laryngeal mask airway (LMA): A tube with a small cushioned mask on the distal end that provides a seal over the laryngeal opening.

laryngoscope: An instrument that is used to displace the tongue and soft tissues, and visualize the larynx and vocal cords during endotracheal intubation.

lidocaine: A medication used to reduce irritation of the mucosal membrane caused by the insertion tube.

lung compliance: The degree of lung expansion per unit pressure change.

lung protection strategy: A method to prevent the lungs from pressure- or volume-induced injuries during mechanical ventilation,

lung-thorax compliance (C_{LT}): The relationship of volume and pressure (V/P) that is imposed by the property of the lungs and thorax. Also called static compliance.

Macintosh blade: a curved laryngoscope blade. Its tip is placed at the vallecula and indirectly lifts the epiglottis for visualization of the vocal cords.

Magill forceps: Special forceps used to perform nasal intubation under direct vision.

Mallampati classification: A method to evaluate the degree of difficulty in intubation.

mandatory minute ventilation (MMV): MMV is a feature of some ventilators that causes an increase of the mandatory frequency (the pressure support level in Hamilton Veolar), when the patient's spontaneous breathing level becomes inadequate. This compensation by the ventilator ensures an adequate minimal minute ventilation.

mass casualty: A large number of severely injured or deaths that exceeds a timely response from regional support centers.

maximum inspiratory pressure (MIP): Also called negative inspiratory force (NIF). MIP reflects a patient's respiratory muscle strength. MIP of less than -20 cm H_2O (e.g., -10 cm H_2O) is one of the indications of impending ventilatory failure. It is obtained by measuring the maximum negative pressure during a forced inspiratory maneuver against a closed manometer.

mean airway pressure (mPaw): The average airway pressure during a complete respiratory cycle. It is directly affected by the respiratory frequency, inspiratory time, peak inspiratory pressure, and positive end-expiratory pressure.

mean arterial pressure: The average blood pressure in the arterial circulation. Normal is >60 mm Hg.

mechanical deadspace: Volume of gas contained in the equipment and supplies (e.g., endotracheal tube, ventilator circuit) that does not take part in gas exchange.

medical futility: A condition in which medical interventions are useless based on past experience.

microprocessor: Minute computer that is designed to perform specific functions.

Miller blade: A straight laryngoscope blade. It lifts up the epiglottis for visualization of the vocal cords during intubation.

monoplace hyperbaric chamber: A hyperbaric enclosure (e.g., cylindrical metal or acrylic crystal clear tube) designed to treat one patient at a time.

multiplace hyperbaric chamber: A large hyperbaric chamber designed to treat more than one patient at a time.

nasal CPAP (N-CPAP): A nasal device for delivering continuous positive pressure to the airway without the need for intubation. The neonate must have adequate and sustainable spontaneous breathing effort to use nasal CPAP.

nasal mask: A mask that covers only the nose.

nasal pillows: Nasal pillows are a small interface commonly used for CPAP therapy.

neurally adjusted ventilatory assist (NAVA): A mode of mechanical ventilation in which the patient's electrical activity of the diaphragm (EAdi or Edi) guides the optimal functions of the ventilator.

nitric oxide: Inhaled nitric oxide (iNO) therapy has been used to treat persistent pulmonary hypertension and hypoxemic respiratory failure of the newborn, respiratory distress syndrome and hypoxemic respiratory failure of older infants and children, and acute respiratory distress syndrome in adults.

nondepolarizing agents: Drugs that compete with acetylcholine for the receptor sites at the motor endplates, thus blocking the normal action of acetylcholine and causing muscle blockade. Examples are vecuronium bromide (Norcuron) and pancuronium bromide (Pavulon).

noninvasive positive pressure ventilation (NPPV): NPPV provides assisted ventilation without an artificial airway.

obstructive sleep apnea (OSA): OSA is caused by severe air flow obstruction during sleep.

oliguria: Below normal urine output.

operative tube thoracostomy: A technique of test tube placement by dissection into the pleura, digital inspection of the pleural space, and insertion guided with the finger and hemostat.

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opioid analgesics: Drugs that are used to control pain via the central nervous system. Examples are morphine, codeine, and meperidine (Demerol, a synthetic compound).

optimal PEEP: The lowest PEEP level leading to the best oxygenation status (or other indicators) without causing significant cardiopulmonary complications.

oronasal mask: A mask that covers the nose and mouth. It is used in noninvasive positive pressure ventilation.

oropharyngeal airway: A device to relieve upper airway obstruction.

oxygen delivery: Total amount of oxygen carried by blood. It is the product of O_2 content and cardiac output.

oxygenation: Amount of oxygen available for metabolic functions; affected by ventilation, diffusion, and perfusion.

oxygenation failure: Failure of the heart and lungs to provide adequate oxygen for metabolic needs.

P/F ratio: PaO_2/F_IO_2. Clinical assessment of oxygenation and degree of hypoxemia, ≤300 mm Hg for ALI, ≤200 mm Hg for ARDS.

pandemic: An occurrence of infectious disease that is spreading through human populations across a large region, continent, or world.

parasympatholytic bronchodilators: Anticholinergic bronchodilators. Drugs that dilate the airways by inhibiting the parasympathetic branch of the autonomic nervous system. Examples are atropine and ipratropium bromide (Atrovent).

peak alveolar pressure (P_{ALV}): Pressure obtained by performing an end-inspiratory pause, also called plateau pressure.

peak inspiratory pressure (PIP): Maximum pressure measured during one respiratory cycle, usually at end-inspiration.

permissive hypercapnia: Intentional hypoventilation of a patient by reducing the ventilator tidal volume to a range of 4–7 mL/kg (normally 10mL/kg). It is used to lower the pulmonary pressures and to minimize the risk of ventilator-related lung injuries. The patient's $PaCO_2$ is significantly elevated and the resulting acidotic pH is neutralized by bicarbonate or tromethamine.

physiologic deadspace: Sum of anatomic and alveolar deadspace. Under normal conditions, it is about the same as anatomic deadspace.

pilot balloon: The small balloon on the proximal end of an endotracheal or tracheostomy tube. It is used to regulate the volume of air in the cuff and to serve as an indicator of air volume in the cuff.

plateau pressure (P_{PLAT}): Pressure obtained by performing an end-inspiratory pause, also called peak alveolar pressure.

pneumatic drive mechanism: Operation of a ventilator with pressurized gas as a power source.

positive end-expiratory pressure (PEEP): PEEP is an airway pressure strategy in ventilation that increases the end-expiratory or baseline airway pressure to a value greater than atmospheric pressure. It is used to treat refractory hypoxemia caused by intrapulmonary shunting.

positive pressure ventilation: Mechanical ventilation in which the volume is delivered by a positive pressure gradient (i.e., airway pressure higher than alveolar pressure).

power: A setting during HFOV that determines the amplitude of oscillation, tidal volume, and degree of ventilation.

preload: The end-diastolic stretch of the muscle fiber.

pressure compensation: A ventilator feature that makes self-adjustment of pressure or volume output based on changing atmospheric pressure.

pressure support ventilation (PSV): A mode of ventilation in which the patient's spontaneous tidal volume is augmented by the application of a preset pressure plateau to the patient's airway during the inspiratory phase of a spontaneous breath.

pressure-controlled ventilation (PCV): Mode of ventilation in which a preset peak inspiratory pressure is used to provide ventilation. The delivered volume during this mode of ventilation is affected by the changing compliance and resistance.

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pressure-regulated volume control (PRVC): A mode of ventilation that provides volume-controlled breaths with the lowest pressure possible by altering the flow and inspiratory time.

pressure-triggered: Initiation of a mechanical breath based on the drop in airway pressure that occurs at the beginning of a spontaneous inspiratory effort.

pressure-volume loop (PVL): Graphic display of changes in pressure and volume during a complete respiratory cycle.

prone positioning (PP): A procedure to temporarily improve a patient's oxygenation by placing the bed and patient in a Trendelenberg position at 15 to 30 degrees.

prophylactic ventilatory support: Early intervention of potential ventilatory failure by means of mechanical ventilation.

propofol (Diprivan): An intravenous drug that provides a spectrum of CNS effects ranging from light sedation to deep general anesthesia.

proportional assist ventilation (PAV): A mode that uses variable pressure to provide pressure support. The variable pressure is in proportion to the patient's pulmonary characteristics (elastance and airflow resistance) and demand (volume or flow).

pulmonary artery pressure (PAP): Pressure measured in the pulmonary artery. It reflects the volume status of the pulmonary artery and the functions of the ventricles. Right ventricular afterload.

pulmonary capillary wedge pressure (PCWP): Pressure measured in the pulmonary artery with a balloon inflated to stop pulmonary blood flow. It reflects the volume status and functions of the left heart. Left ventricular preload.

pulmonary vascular resistance: Resistance of the arterial system into which the right heart is pumping. Normal range is 50–150 dynes.sec/cm^5.

pulse contour analysis: A less invasive method to calculate the stroke volume and stroke volume index by using the area under the arterial pressure waveform and specific patient data.

pulse oximeter: A device that estimates arterial oxygen saturation (SpO_2) by emitting dual wavelengths of light through a pulsating vascular bed.

radiopaque: Impenetrable to X-rays. It appears as a light area on the radiograph.

Ramsay Scale: The scoring system with a scale ranging from level I to level VI; used to assess the degree of sedation.

rapid sequence intubation (RSI): Intubation with an endotracheal tube under controlled conditions.

rapid shallow breathing index (RSBI): The RSBI (f/V_T ratio) is calculated by dividing the spontaneous breathing frequency (breaths/min) by the average spontaneous V_T (L). Absence of rapid shallow breathing, as defined by an f/V_T ratio of less than 100* breaths/min/L, is an accurate predictor of weaning success. (*rounded from 105 breaths/min/L.)

refractory hypoxemia: A persistent low level of oxygen in blood that is not responsive to medium to high concentration of inspired oxygen. It is usually caused by intrapulmonary shunting.

renal perfusion: Blood flow to the kidneys. It is decreased when blood volume or cardiac output is low.

SALT: A mass casualty triage algorithm consists of Sort, Assess, Life-saving interventions, and Treatment/Transport.

servo: A feedback system that typically consists of a sensing element, an amplifier, and a servomotor, used in the automatic control of the mechanical device of a ventilator.

sine wave: A graphic presentation of flow and time that has a horizontal "S" appearance.

sniffing position: An ideal head position for endotracheal intubation. It is done by tilting the forehead back slightly and moving the mandible anteriorly to the patient.

SOFA: The Sequential Organ Failure Assessment (SOFA) score is a triage system that uses six criteria to predict the outcomes of critically ill patients in the hospital.

solenoid valves: A valve controlled by an electronic switching device that is used to regulate the specific functions of a ventilator.

speaking valve: A one-way valve attachment to the tracheostomy tube that allows the user to talk.

spontaneous breathing trial (SBT): An evaluation of a patient's readiness for weaning from mechanical ventilation and extubation. Spontaneous breathing may be augmented with a low level ($\leq$8 cm H_2O) of pressure support, CPAP, or automatic tube compensation (ATC). SBT may last up to 30 minutes.

spontaneous ventilation: Volume of gas inspired by a patient. It is directly related to the patient's spontaneous tidal volume and frequency.

START: The Simple Triage and Rapid Treatment algorithm that is based on three parameters: respirations, perfusion, and mental status (RPM). The pediatric version is known as JumpSTART.

strategic national stockpile: A federal program that stores large quantities of medicine and medical supplies in a centralized location for the public in case of a health emergency.

stroke volume: Blood volume output delivered by one ventricular contraction.

stylet: A flexible but semirigid wire placed inside an endotracheal tube to provide a desired curvature for oral intubation.

subglottic secretion drainage: This procedure uses a special endotracheal tube with a separate dorsal lumen for suctioning of secretions above the ET tube cuff.

surfactant: A natural phospholipid that lowers the surface tension of the lungs. Deficiency of surfactant causes high surface tension in the lungs and increases the work of breathing.

surfactant replacement: Direct instillation of synthetic surfactant (Surfaxin) or natural surfactant (Survanta, Infasurf) into the trachea.

sympathomimetic bronchodilators: Adrenergic agonists. Drugs that dilate the airways by stimulating the beta-2 receptors of the sympathetic nervous system. Examples are epinephrine (Adrenaline) and albuterol (Ventolin, Proventil).

synchronized intermittent mandatory ventilation (SIMV): SIMV is a mode in which the ventilator delivers control (mandatory) breaths to the patient at or near the time of a spontaneous breath. The mandatory breaths are synchronized with the patient's spontaneous breathing efforts to avoid breath stacking.

systemic vascular resistance: Resistance of the arterial system into which the left heart is pumping. Normal range is 800–1,500 dynes.sec/cm^5.

thoracic pump mechanism: Alternations in pulmonary blood flow caused by changes in intrathoracic pressure during positive pressure ventilation. In hypotensive conditions, positive pressure ventilation decreases the blood flow to the left heart. In hypertensive conditions, this mechanism enhances the outflow of blood from the right ventricle and into the left heart.

three-chamber drainage system: A chest tube drainage setup that requires a vacuum source to provide continuous suction.

tidal volume (V_T): Volume delivered by ventilator during mandatory breaths.

time-triggered: Initiation of a mechanical breath based on the set time interval for one complete respiratory cycle (inspiratory time and expiratory time).

total cycle time (TCT): Time period from beginning inspiration to the end of expiration (beginning of next inspiration).

total parenteral nutrition (TPN): Complete nutritional support provided to the patient by any method (usually intravenous) other than the intestinal route.

tracheal gas insufflation (TGI): Use of a small catheter to provide a continuous or phasic gas flow directly into the trachea during mechanical ventilation.

tracheotomy: A surgical procedure to create an opening into the trachea.

tracheostomy tube: An artificial airway inside the trachea that is inserted through a surgical opening at the trachea.

transairway pressure (P_{TA}): Flow-resistive pressure, the difference between airway opening pressure (P_{AO}) and alveolar pressure (P_{ALV}), or $P_{TA} = P_{AO} - P_{ALV}$.

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transbronchial lung biopsy (TBLB): Tissue specimens collected by the forceps during a forced exhalation maneuver.

transbronchial needle aspiration biopsy (TBNAB): Tissue specimens collected by applying aspiration while moving the needle at the sample collection site.

transcutaneous PCO_2 ($P_{tc}CO_2$): Measurement of PCO_2 through the skin by means of a miniature Severinghaus (PCO_2) electrode.

transcutaneous PO_2 ($P_{tc}O_2$): Measurement of PO_2 through the skin by means of a miniature Clark (PO_2) electrode.

transesophageal echocardiography: A method using a Doppler transducer in the esophagus for an indirect measurement of the blood flow velocity in the descending aorta and the calculation of the cardiac output and other hemodynamic data.

transport ventilator: A mechanical ventilator capable of operation without piped-in gas sources or electrical connection.

transtentorial herniation: A type of brain injury that causes the downward displacement of the medial aspect of the temporal lobe (uncus) through the tentorial notch by a mass above.

triage: A process that uses predetermined criteria to assign individuals from a large pool of people for grouping and making decisions.

trocar: A sharply pointed instrument for incision into the chest cavity.

trocar tube thoracostomy: A technique of chest tube placement by incision into the pleura, insertion of trocar chest tube, and withdrawal of trocar.

unplanned extubation: Unexpected removal of an endotracheal or tracheostomy tube before the patient is ready for extubation.

V/Q mismatch: An abnormal distribution of ventilation and pulmonary blood flow. High V/Q is related to deadspace ventilation, whereas low V/Q is associated with intrapulmonary shunting.

vagus nerve: The pneumogastric or tenth cranial nerve. Its superior and recurrent laryngeal nerves and their branches adjoin the upper end of trachea and are sensitive to stimulation by the endotracheal tube or suction catheter.

vallecula: An area between the base of the tongue and epiglottis; an anatomical landmark for the placement of the curved (Macintosh) blade.

venous return: Blood flow from the systemic venous circulation to the right heart.

ventilation/perfusion (V/Q) mismatch: An abnormal distribution of ventilation and pulmonary blood flow. High V/Q is related to deadspace ventilation, whereas low V/Q is associated with intrapulmonary shunting.

ventilator-associated pneumonia (VAP): A severe form of hospital-acquired infection of the lung parenchyma that develops within 48 hours after intubation and initiation of mechanical ventilation.

ventilatory failure: Failure of the respiratory system to remove CO_2 from the body resulting in an abnormally high $PaCO_2$.

vocal cords: Two thin, almost parallel folds of tissue within the larynx that vibrate as air passes between them; an important landmark as the entry point to the trachea during intubation.

volume ventilation plus (VV+): An option that combines volume control plus and volume support.

volume-assured pressure support (VAPS): A mode of ventilation that assures a stable tidal volume by incorporating inspiratory pressure support ventilation (PSV) with conventional volume-assisted cycles (VAV).

volume-controlled ventilation (VCV): Mode of ventilation in which a preset tidal volume is used to provide ventilation. The airway pressures during this mode of ventilation are affected by the changing compliance and resistance.

weaning failure: Failure of spontaneous breathing trial (SBT) or the need for reintubation within 48 hours following extubation.

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weaning in progress: An intermediate category (between weaning success and weaning failure) for patients who are extubated but continue to receive ventilatory support by noninvasive ventilation (NIV).

weaning success: Absence of ventilatory support 48 hours following extubation.

xanthine bronchodilators: Drugs that produce bronchodilation by inhibiting phosphodiesterase, an enzyme that inactivates cyclic 3'5' AMP (a substance that promotes bronchodilation). Examples are oral theophylline (Theo-Dur, Slo-bid) and aminophylline, a water-soluble theophylline (Aminophyllin, Somophyllin).

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